BRAIN MATURATION AND COGNITIVE DEVELOPMENT

Comparative and Cross-Cultural Perspectives

Edited by

Kathleen R. Gibson and Anne C. Petersen

Sponsored by the Social Science Research Council

ALDINE DE GRUYTER
New York
1991

Chapter 8

Universals of Behavioral Development in Relation to Brain Myelination

Melvin Konner

Introduction

Pediatrics, neurology, and psychiatry assume that key elements of normal psychological and behavioral development are understood (Rudolph and Hoffman, 1982, Chap. 4; Adams and Victor, 1981, Chap. 27; Nicholi, 1978, Chap. 23). In part, these assumptions form the basis for clinical assessment and treatment, including recommendations about primary prevention that are staples of pediatric and psychiatric advice. Some presumed "facts" of early development, however, are based on minimal data. For example, some clinicians believe that immediate postnatal contact between mother and infant is essential for "mother infant bonding" (Klaus and Kennell, 1976), but the evidence for this assertion is at best very weak (Svejda et al., 1982). Similarly, many Western clinicians believe that infants should outgrow night-waking after about 3 months of age and assume that this transition reflects patterns of neurological maturation (Spock, 1976). Elaborate treatment programs may be instituted to extinguish night-waking after this time. Many of the world's cultures, however, consider night-waking to be normal and expectable until as late as 3 years of age and make no attempt to extinguish it at earlier ages (Konner and Worthman, 1980).

Recently, doubt has even been cast on one of the most fundamental beliefs about human psychological development (Kagan et al., 1978; Kagan, 1984). A substantial body of opinion holds that the importance of infancy has been greatly exaggerated. Despite much evidence from animal models showing lasting effects of early experience, decisive clinical evidence of comparable effects in humans has been difficult to find. Early experience has not been clearly shown to have lasting important, or even measurable, effects on the course of psychological development. This recent viewpoint, if true, must jeopardize some of what is now common clinical practice in psychiatry and behavioral pediatrics.

Cross-cultural and cross-population studies of psychological development have had, as a main purpose, the demonstration of flexibility of the human psychological repertoire and, by inference, of the underlying neural and endocrine functions. Few studies have attempted to delineate cultural universals of development. Yet, it is increasingly recognized that much of psychological development is inflexible in the face of marked variation in environment of rearing (e.g., Eibl-Eibesfeldt, 1971a,b, 1983; Kagan, 1976; Konner, 1982; Super, 1981).

Developmental studies have also lacked a clear delineation of the underlying events of regional neurological and/or neuroendocrine development. In past decades, this lack was generally attributed to the absence of adequate studies of structure–function relationships and of brain development itself, particularly in humans, and, particularly, in the late prenatal and early postnatal periods. Recent work, however, has produced great advances in relevant fields, including neuroanatomy (e.g., Nauta and Domesick, 1980), neurophysiology (Evarts, 1975; Waxman, 1977, 1982), behavioral neurology (Heilman and Valenstein, 1979; Geschwind and Galaburda, 1985), neuroethology (MacLean, 1978), and developmental neurology (Brody et al., 1984; Geschwind and Galaburda, 1985; Gilles et al., 1983; Yakovlev and Lecours, 1967). We now know that changes in brain structure and function during the first few years of postnatal life are rapid and large. In the first year of life the brain more than doubles in volume, reaching 60% of its adult size. Growth rate declines only gradually during the second year (Yakovlev, 1962; Blinkov and Glezer, 1968). This high rate of postnatal volumetric increase in brain tissue may be unique to our species among the higher primates and has been viewed as one of the most distinctive advances of human evolution (Gould, 1977).

In the course of this growth, profound structural changes occur in the nervous system. Some basic processes are almost complete by birth. Most neurons, for instance, have already formed, and their cell bodies have migrated to their destined places. Other processes, however, proceed in extrauterine life: proliferation of synapses, branching of dendrites, changes in the density of dendritic spines, changes in connectivity, proliferation of glial support cells through cell division, and the formation of myelin sheaths around axons in the white matter of the central nervous system (Jacobson, 1978). These processes involve attendant and/or underlying neurochemical changes. They are influenced by experience, but to a large extent proceed independently of experience. Each year a considerable literature increases our understanding of these developmental events.

Advances in our knowledge of behavioral development have paralleled those of neural development. Behavioral studies have become increasingly systematic and precise in their methods of measurement and have taken advantage of modern computers to analyze large volumes of behavioral data in an unprecedentedly sophisticated way (Osofsky, 1979; Field et al., 1981; Mussen, 1983). Most behavioral studies, however, fail to consider neurological development.

The persistent lag in incorporating advances in neuroscience into explanations of early psychological development may result from the difficulty of mastering both the language of behavioral development and that of neuroscience, as well as from the reluctance of some students of behavioral and psychological development to come to terms with the existence and nature of fixed maturational sequences. Such sequences have long been known and accepted in the realm of motor development, and have become increasingly established in the realm of cognitive and social development.

This chapter reviews evidence for fixed sequences of behavioral and psychological development drawing on cross-cultural and cross-population studies throughout the world, and relates these behavioral data to brain development in infants and children. Based on these data, a model is proposed to account for certain universals of psychological behavioral development in infancy in terms of maturing brain function.

Assessment of Behavioral and Neuroanatomical Development

Assessment and Observation of Infant Behavior

During the 1960s and 1970s, new methods of assessment led to rapid advances in the study of infant behavior. Anthropologists, cross-cultural psychologists, pediatricians, and psychiatrists readily adopted these methods and applied them to populations throughout the developing world. The result is a new body of knowledge pertaining to cross-cultural and cross-population maturational variations and constancies. This chapter draws on data derived from several of these assessment methods including tests of neurological maturation, motor development, and social development.

Neurological and Behavioral Assessment of Neonates. Two neurological assessment techniques widely used in cross-cultural studies are the Prechtl procedure (Prechtl and Beintema, 1964; Beintema, 1968) and the Brazelton Behavioral Assessment Scale (BNBAS; Brazelton, 1973).

The Prechtl procedure is rigidly standardized and controls for behavioral state of the infant, time since last fed, nonneurological health status, age in days, order of examination, and other variables. The infant is numerically scored on more than 150 assessment items, including behavioral observations, color, muscle tone, joint mobility, a wide variety of specific signs (e.g., hiccups and cough) and, in particular, a comprehensive array of neurological reflexes, both normal (e.g., the Moro reflex elicited by three different stimulus procedures) and abnormal (e.g., Chvostek's sign). The examination generally takes about an hour and requires some training to learn. This procedure has satisfactory interobserver and test–retest reliability if done after 3 days of age with proper attention to state variables.

The Brazelton Neonatal Assessment Scale (BNBAS; Brazelton, 1973) consists of 27 subscales on which the infant is scored from one to nine, with each level defined in detail. The subscales include such items as "alertness," "irritability," and "hand-to mouth facility." Despite the greater difficulty of obtaining agreement on such measures, this scale has also proved to have satisfactory interobserver and test–retest validity after proper training.

Assessment of Motor Development. Cross-cultural data on motor development derive primarily from four tests: the Bayley scale of infant development (Bayley, 1965, 1969), a similar test used in Britain (Griffiths, 1954), the Gesell scales (Gesell and Amatruda, 1947; Knobloch, 1958), and the McGraw scale (McGraw, 1943). Bayley norms exist and have been validated for large samples of both the white and the black population in the United States. The Griffiths test has been less well validated, but has been more frequently used throughout the British colonial network. The Gesell scales leave much to be desired in terms of standardization but are acceptable for crude comparison. Although standardized on much smaller samples, the McGraw scale renders a more sophisticated assessment of motor development than is possible with the other scales. Rather than assessing broad developmental milestones, the McGraw scale follows the gradual emergence of specific motor skills such as sitting or walking throughout infancy.

Observation and Assessment of Social Development Systematic, timed observations with standardized settings and clearly defined codes for the recording of behavior make reliable and valid measurements of social development possible. Modern electronic recording equipment and computers make possible the collection and analysis of unprecedented volumes of behavioral data, and remove much of the subjective even from categories of behavior such as social interaction. Some items of social behavior, such as smiling and crying, can be assessed and recorded with as much or greater accuracy than neurological reflexes or motor milestones.

Such methods of observation and analysis have been frequently reviewed (e.g., Whiting and Whiting, 1975; Cairns, 1974). In the typical study a preset sample of the infant's time (usually 15–90 min) is recorded in a predetermined setting (e.g., with mother in nonfeeding interaction) and with a list of behaviors of established observational reliability. Initial state is usually defined as alert, not hungry, not irritable, and free of illness, among other constraints. In the best studies, repeated samples of the infant's time minimize transient variation, and, ideally, the order of observations is generated from a random number table. Analysis may include calculation of frequency and duration of events, contingency of some events on others, lag sequence analysis designed to reveal complex causal interaction chains, and other approaches.

Cross-population comparisons using these methods of observation must carefully control for cultural variations in setting, response to observations, and other extraneous sources of variance. In any case, these methods represent a great technical improvement over the methods of two or three decades ago, and are greatly superior to the methods of screening for social maturation in common use in pediatric practice.

Specific tests of social responsiveness are numerous and varied. Three tests that have been applied cross-culturally figure in the model of social development presented here: Gewirtz's test of the development of social smiling (Gewirtz, 1965), tests of the development of stranger reactions (Ricciuti, 1974; Konner, 1972; Chisholm, 1983), and Bretherton's and Ainsworth's test for the infant attachment (1974). Gerwirtz's method appraises the development of social smiling in early infancy by beginning with the infant in a quiet alert state and presenting the face of a live, but impassive, adult in a face-to-face juxtaposition with that of the infant. The number of smiles seen in a period of 1 min is recorded.

Ricciuti (1974), Konner (1972), Chisholm, (1983) and others appraise the development of stranger reactions during the period after 6 months of age using minor variations of the following paradigm. The infant or toddler is sitting near the mother and is approached slowly, in a nonthreatening way, by a completely unknown adult who often speaks in a designated manner intended to evoke a positive reaction. Specific behaviors both positive and negative are recorded, including, among others, smiling, approaching the stranger, gaze aversion within 2 sec, withdrawing, and crying. Positive responses are summed arithmetically or weighted according to intensity of response (i.e., more points for crying than for gaze aversion). The sum provides a score for a given infant at a given age in response to this stimulus.

Bretherton and Ainsworth's (1974) test for the evaluation of attachment involves a defined set of behaviors of the infant toward the mother or other primary caretaker in a novel situation. Briefly, the infant and mother are brought into a room with toys on the floor. The infant is given 3 min to acclimatize to the room, whereupon a stranger enters, chats with the mother, and interacts with the infant for 1 min each. The mother then leaves the room for 3 min, or until the infant cries for 30 sec, and returns for 3 min with the stranger absent. Similar staged events, including leaving the infant alone, follow for a total of eight episodes. The infant's behavior is recorded according to a predetermined list of items, with an emphasis on crying and other protest behavior on mother's departure, and on behavior at reunion.

Myelination and Myelination Cycles

Myelin Structure and Function. Myelin, apparently first named by Virchow in 1864, is a fatty sheath that insulates a large proportion of mammalian nerve fibers, both peripheral and central. This insulation is a space saving and energy efficient adaptation that provides for rapid impulse conduction without necessi

lating the great fiber size characteristic of rapidly conducting fibers in invertebrate nervous system.

The composition of myelin is grossly similar to that of typical cell membranes, but with a higher ratio of lipid to protein and a larger proportion of glycolipids relative to phospholipids and cholesterol. The proteins, embedded in the lipid bilayer in a manner common in other plasma membranes, include, among others, myelin basic protein, proteolipid protein, and glycoprotein and enzymes (Peters et al., 1976; Raine, 1984a).

Myelination Sequences in Development. Since the introduction of the Weigert stain in the late nineteenth century, myelin-stained sections of brain and spinal cord have been a mainstay of neuroanatomic research and teaching. The Weigert stain and its successors, the Loyez stain (Yakovlev, 1970) and the Luxol fast blue stain, are relatively easy to make and to interpret. Thus they permit easy visualization and tracing of fiber pathways.

For example, the projection of optic radiation from the lateral geniculate body of the thalamus to area 17 of the occipital cortex becomes stainable (acquires myelin) during the immediate postnatal months in the human brain, making it stand out at that time from the background of unstained (unmyelinated) pathways in most other parts of the cortical radiations. The vivid stainability of the myelin sheath together with the regionally specific timing of myelination made studies of Weigert-stained sections of fetal and young postnatal brains of various species a mainstay of neuroanatomical tracing in the era when neural circuits were first being delineated.

In the peripheral nervous system, myelin is the product of the Schwann cell, while in the central nervous system it is produced by another glial cell type, the oligodendrocyte. Under the light microscope, the developmental process of myelination has several distinct phases (Yakovlev and Lecours, 1986; Gilles et al., 1983). Some time after the extension of axions toward or, more likely, to their sites of termination, marked glial cell hyperplasia occurs in the vicinity of the axon. This process has been termed "myelination gliosis" by analogy (technically inappropriate) with gliosis that occurs in the vicinity of injured neurons. The glial cells so generated accumulate myelin lipid components ("premyelin lipids") cytoplasmically before the actual appearance of myelin. Following myelination gliosis there are at least three recognizable phases of myelination in appropriately stained sections: (1) myelin visible only microscopically, (2) myelin visible to the naked eye, but faintly in comparison to the ultimate or mature level, and (3) mature level of myelin density.

Although the progress from myelination gliosis to grossly visible myelin is complete in a matter of weeks in most systems, the attainment of the mature level of density may take months or even years in some systems. Central nervous system pathways, in particular, myelinate at distinctly different times during development, and despite some significant timing differences along the course of a given pathway (a cephalocaudal direction of myelin deposition seems to be a

regular feature), there are greater timing differences between than within pathways.

Myelination as an Index of Regional Brain Development. Originally, myelination studies focused on the more central purpose of using immature brains to illuminate connections. Ever since the pioneering work of Flechsig (1920), however, myelination has been used to illuminate developmental sequences and attempts have been made to correlate myelination and behavioral maturation. These attempts have engendered some controversy and criticism because the interrelationships between myelination, maturation, and behavior are not transparently obvious.

Encyclopedic treatments of the development of the human cortex by Conel (1939–1967) and his successor, Rabinowicz (1979), however, have shown that various aspects of cortical development exhibit considerable synoptic growth: e.g., width of the cortex and its layers; number, size, and density of neurons; condition of intercellular components such as chromophil substance and neurofibrils; number and size of extrinsic fibers; and number, size, and form of neuronal processes, including the density of "pedunculated bulbs" (now called thorns or spines) on the dendrites. These data suggest the validity of myelination sequences as a crude general index of regional brain development. A rank ordering of the relative maturity of a series of cortical regions at a given age would be quite similar whichever of Conel's indices were used. Although Conel's work has been criticized for methodological inadequacies (Purpura and Reaser, 1974), these inadequacies appear to affect the materials he studied uniformly, so that at worst the timing, but not the sequence, of developmental events may have been inaccurately described. The consistency of rank order should and does survive uniformly expressed difficulties of method.

Experimental animal studies also demonstrate correlations between myelination sequences and other indices of regional brain development. Numerous changes occur in cell bodies during myelination of their axons. For instance, quantities of cytoplasm and axoplasm increase in direct proportion to each other and changes occur in neuronal packing density, nuclear and nucleolar diameters, and distribution of chromophil substance within the cell (Martinez and Friede, 1970). In addition, the extent of myelination correlates with the growth of axon diameter, so much so that axon diameter has been proposed as the trigger for myelination (Friede and Samorajski, 1967; Matthews, 1968), although this view may be oversimplified (Moore et al., 1976). It thus appears that myelination sequences can serve provisionally as a crude index of regional brain development.

Myelination and Nerve Function. Whether myelination can serve as an index of behavioral capability is a separate issue. Early investigators thought that it could (Tilney and Casamajor, 1924; Windle et al., 1934; Keene and Hewer, 1931; Langworthy, 1933). It is now clear, however, that myelination cannot be

considered "an absolute index of behavioral capability" (Angulo y Gonzalez, 1929).

In both rats and humans, neurological function begins before myelin appears. In many species critical functions in the adult, such as those of the autonomic nervous system, occur in normally unmyelinated fibers. Many invertebrate nervous systems function without myelin, and anatomically specific demyelination does not invariably result in the expected loss of function in clinical syndromes such as multiple sclerosis. For these reasons, some modern authors consider myelination sequences irrelevant to the development of behavior (e.g., see Kinsbourne and Hiscock, 1983, with special reference to the role of myelination of the cerebral commissures in the development of lateralized hemispheric function).

Such cautionary remarks have validity, but they represent only one side of a complex set of questions about the role of myelination in nerve function. On the other side are arrayed a large body of clinical data from the study of a variety of syndromes involving demyelination or delayed myelination, mounting evidence from experimental animal models of demyelination and delayed myelination, and theoretical and experimental considerations relating to membrane function with and without myelin, all of which support a significant, if imperfect, relationship between myelination and function (Ritchie, 1984; Waxman, 1977, 1982).

Nerve fibers subjected to a pathological or experimentally induced loss of myelin share certain functional alterations. These include (1) decreased conduction velocity, (2) increased refractory period, (3) more frequent conduction failure, (4) temporal dispersion of impulses, (5) increased susceptibility to inadvertent electrical modification by neighboring axons, and (6) increased susceptibility to mechanical, thermal, and other extraneous influences (McDonald and Sears, 1970; Rasminsky and Sears, 1972; Ritchie, 1984; Waxman, 1977, 1982). These effects are listed in Table 1, and some of the studies demonstrating them are listed in Table 2.

In addition, studies of remyelination have demonstrated convincing correlations between reacquisition of myelin and reappearance of normal or approximately normal conduction latency and refractory period in the remyelinating fibers (Smith, et al., 1981).

It is not clear, however, that evidence about demyelination or remyelination, even when consistent across different clinical and experimental models, can be transferred to the normally unmyelinated condition. There may be abnormalities of the membrane underlying damaged or diseased myelin that do not characterize the membrane of developing not-yet-myelinated cells. The few existing studies of functional consequences of lack of myelin in normal systems appear to corroborate these findings, however.

In the best study of this nature, Huttenlocher (1970) followed the developing cat pyramidal tract from 3 to 5 weeks of postnatal life and found that several functional capabilities precede myelination. The ability of the fiber to conduct

Table 1. Consequences of Demyelination[a]

Increased conduction latency (up to 30×)
Increased refractory period
Impairment at higher frequencies
Conduction block more likely
Temporal dispersion of impulses
Ephaptic communication ("cross-talk")
Temperature and mechanical sensitivity

[a] From Ritchie (1984) and Waxman (1977, 1982).

Table 2. Correlates and Consequences of Myelination and Demyelination

Study	*Model System*	*Finding*
Selected landmark studies		
Huxley and Stämpfli (1949)	Frog myelinated nerve fiber	First demonstration of saltatory conduction, restriction of ion flow to node of Ranvier
McDonald and Sears (1970)	Cat dorsal column, diphtheria toxin demyelination	Decreased conduction velocity, increased refractory period, high frequency block
Rasminsky and Sears (1972)	Rat ventral root, diphtheria toxin demyelination	Internodal conduction time increased from 20 to 600 μsec
Rasminsky (1973)	Rat ventral root, diphtheria toxin demyelination	Exquisite temperature sensitivity (block with increase of 0.5°C)
Raminsky (1980)	Dystrophic mice, spinal nerve roots	Ephaptic transmission ("cross talk") among single fibers
Developmental studies		
Huttenlocher (1970)	Cat pyramidal tract, 3 days to 5 weeks	Myelination makes repetitive firing possible
Martinez and Friede (1970)	Rat sciatic nerve, 1–16 weeks	Multiple correlates in axon and cell growth
Freeman (1978)	Cat optic nerve, adult, no treatment	Myelin sheath thickness predicts conduction latency in mature fibers
Recovery studies		
Smith et al. (1981)	Cat dorsal, column, adult, LPC demyelination	Remyelination restores experimentally blocked conduction
Bostock et al. (1981)	Cat dorsal column, adult, LPC demyelination	Conduction precedes remyelination due to nodal aggregation of Na^+ channels

impulses under the stimulus of a high-frequency train, however, was absent before myelination. Rates of repetitive firing as low as 40/sec resulted in conduction block. Since rates of 50–100/sec have been shown to be involved in the normal course of pyramidal tract function during voluntary contraction of hand and forearm muscles in monkeys, the limitations of premyelinated neurons would have functional significance. Metabolic considerations support this interpretation. The active membrane surfaces of unmyelinated axons is two to three orders of magnitude greater than that of myelinated axons. The energy expenditure required to maintain the same rate of firing in the unmyelinated condition would be formidable.

Experience Effects on Myelination. Although much is known about the events leading up to and associated with early myelination (Peters et al., 1976; Raine, 1984a; Sidman and O'Gorman, 1981), there is not yet a convincing account of what causes it. Possibly, function not only precedes but also causes myelination. This hypothesis is consistent with findings that experience or exercise affect brain development. Early experience, for instance has dramatic effects on the structure and function of neurons in the visual cortex of cats and monkeys (Wiesel and Hubel, 1965; LeVay et al., 1980). Although less dramatic, consistent effects have been demonstrated of environmental enrichment on the thickness, neuronal density, dendritic branching complexity, dendritic spine counts, synaptic density, acetylcholine level, cholinesterase activity level, and glia-to-neuron ratio in the cerebral cortex of the rat (Globus et al., 1973; Diamond et al., 1964, 1985).

Studies of the effects of experience and exercise on myelination have not been as numerous or impressive as these classic studies, but they have shown a moderately consistent effect of experience, as indicated in Table 3 (Gyllenstein and Malmfors, 1963; Wendell-Smith, 1964; Kingsley et al., 1970; Moore et al., 1976; Sammeck, 1975; Samorajski and Rolsten, 1975; Tauber et al., 1980).

Although these studies show that there is a significant effect of experience on myelination, these effects are either transient or, if permanent, relatively small - on the order of 10–20%. The most severe procedures of stimulus deprivation, such as rearing in darkness, total occlusion of an eye, or sciatic neurectomy, are compatible with the eventual acquisition of normal myelin in the great majority of fibers in the deprived nerves or tracts. This strongly suggests an underlying process whose timing is under genetic control.

A considerable literature supports this suggestion. Among the infantile diseases of the cerebral white matter at least six, known as dysmyelinating leucodystrophies and resulting in specific retardation syndromes, have been shown to have simple Mendelian inheritance patterns (Carter and Gold, 1974; Raine, 1984b; Traugott and Raine, 1984). Clinical syndromes of specifically peripheral hypomyelination in infants suggest a genetic basis as well. These syndromes are characterized by global delays in motor development that are unresponsive to exercise and consistent with the delay or insufficiency of myelination (Ono et al., 1982).

Table 3. Experience Effects on Myelination

Study	*Model System*	*Finding*
Gyllensten and Malmfors (1963)	Mice reared in darkness, birth to 30 days	12% reduction in number of myelinated fibers in optic nerve
Wendell-Smith (1964)	Mice, one eye occluded, birth to 75 days	"Visual impression" of decreased sheath thickness in optic nerve
Kingsley et al. (1970)	Rats, sciatic neurectomy, birth to 21 days	10–20% reduction in number of myelinated fibers in dorsal funiculus
Sammeck (1974)	Rats, swimming 2–4 h/day, from "adolescence" for 12–16 days	"Considerable increase" in number of myelinated axons in sciatic nerve
Samorajski and Rolsten (1975)	Mice, activity wheel running 2–4 h/day from 3 to 24 months	No effect on posterior tibial nerve myelin despite fiber hypertrophy
Moore et al. (1976)	Cats reared in darkness, birth to 4 weeks	No effect on percentage of myelinated axons
Tauber et al. (1980)	Rabbits, artificial eye opening at 5 days (vs. 10 days)	Myelin-specific proteins double at 7–10 days, but equal at 20 days

Animal models have been more directly supportive of a primarily genetic control of myelination, since they permit deciphering some of the details of development control (Hogan and Greenfield, 1984; Carnow et al., 1984). For example, two mutants of the mouse affecting myelination—"jimpy," an X-linked mutation that causes severe CNS hypomyelination while sparing the PNS, and "quaking," an autosomal recessive allele on chromosome 17 causing global hypomyelination—have been studied with respect to myelin basic protein (MPB) gene expression. Messenger RNA transcription is normal in both cases, but MPB-related translation products are altered in the proportions of different molecular weight species that become particularly relevant at different stages of myelin synthesis (Carnow et al., 1984). Such studies, carried out in parallel with continued ultrastructural examination of the myelination process, should lead to a general characterization of the genetic control of myelination in the not too distant future.

Myelination, then, whether pre- or postnatal, must be considered to be a genetically controlled process largely intrinsic to the growth and differentiation of the brain. Its influence on developing behavior is likely to be large compared with the reciprocal influence of experience on myelination, although the latter is certainly significant, and mutual interaction of biology and experience must always be considered to be the rule.

Human Myelination Data. Paul Flechsig (1920) was the first to provide detailed descriptions of the sequence of myelination in humans. His reputation is

closely associated with the mapping of the sequence of myelination within the cerebral cortex. During the 1920s and 1930s, Flechsig's work inspired a number of studies directed toward correlating myelination sequences with the development of behavioral capability, particularly during fetal development (Tilney and Casamajor, 1924; Windle et al., 1934; Keene and Hewer, 1931; Langworthy, 1933).

In the modern period, several investigations have expanded and confirmed the work of Flechsig. Yakovlev and Lecours (1967) studied a total of 200 brains, ostensibly neurologically normal, ranging from early gestation to late in the senium. The unique value of their series is that all the brains were uniformly preserved, cut, and stained.[1] The brains were fixed with formalin, imbedded in celloidin, and serially sectioned at 20–40 um. Every tenth section was then stained according to the Loyez modification of the Weigert hematoxylin stain for myelin sheaths, and the adjacent section stained with the Bielschowsky–Plien cresyl violet stain for cell bodies. (All details of method are given in Yakovlev, 1970.) Yakovlev and Lecours (1967) then studied the myelin-stained sections with the light microscope using nonquantitative methods of description and comparison. The essence of this method is the preservation of whole brain serial sections giving the opportunity to reconstruct the main elements of three-dimensional brain anatomy, including all circuitry distinguishable with the myelin stain.[2]

Rorke and Riggs (1969) conducted a study specifically directed to the perinatal status of the brains of infants of varying birthweight (from 740 to 3910 g) to represent a range of gestation lengths from quite premature to full-term. The entire sample consisted of 107 infants of which 23 were considered to be full-term and normal sized. Death occurred within 7 days of birth (96% were either stillborn or died within 3 days), and was, in most cases, a result of hyaline membrane disease, pneumonia, or pulmonary hemorrhage. After celloidin-embedding, a limited but representative number of sections (around 30) of each brain were taken at 20 um and stained with luxol fast blue and cresyl violet according to the technique of Klüver–Barrera. As was the case in the Yakovlev and Lecours study, assessment was made by careful visual inspection and description rather than by morphometric or other quantitative methods. These authors were the first to introduce the refinement of recognizing and describing individual variation at a given age. However, for the ostensibly full-term normal-sized infants, 87% of the specimens showed "essentially similar" myelination, with the other 13% showing lesser degrees of development.

Gilles et al. (1983 see Table 4) conducted their study of myelination as part of the National Collaborative Perinatal Project (NCPP) of the National Institute of Neurological and Communicative Disorders and Strokes. They used 323 cases from NCPP collection, most of which were Loyez stained (see above), ranging in estimated age from 20 to 48 postconceptual weeks. They introduced a number of refinements of method compared with prior investigators. First, they declined to

Table 4. Comparison of Several Estimates of Ages of Onset of Myelination in the Human Fetal Nervous System[a]

	Gilles, et al. 1983 (Microscopic Myelin)	*Yakolve and Lecours (1967)*	*Larroche (1966)*	*Langworthy (1933)*
Spinal cord				
Fasciculus gracilis	< 20	—	28	28
Dorsal spinocerebellar	< 20	—	26	20
Spinothalamic	< 20	—	26	28
Brainstem				
Medial longitudinal fasciculus	< 20	20–22	24	20
Medial lemniscus	23–25	24	26–28	28 and 36
Acoustic	23–24	20–24	24–28	38
Inferior cerebellar peduncle	24	24	28	
Superior cerebellar peduncle	26	32	32	28
Corticospinal	32–35	38	28–36	4 PN
Transpontine	35	4 PN		—
Middle cerebellar peduncle	35	8 PN	PN	4 PN
Cerebellum				
Parasagittal	27	—	32	—
Hemispheral	38	PN	40	
Prosencephalon				
Habenulointerpeduncular	28	28	34	28
Ansa lenticularis	28	28	26	
Optic chiasm	32	—	32	—
Optic tract	29	36	32	36
Optic radiation	38–39	38–40	38	—
Internal capsule, posterior limb	32	36	36	
Corona radiata	34	30	—	
Corticopontine	Latl. ped.–38 Medl. ped.–46	8 PN	—	8 PN
Cingulum	38	8 PN	—	
Fornix	39	16 PN	PN	8 PN
Callosum	46	12 PN	PN	4 PN
Anterior commissure	46	12 PN	—	—
Mammilothalamic	48	36	—	8 PN

[a]All figures are in weeks of gestational age or postnatal age (PN) as indicated by each author. This table is reproduced with permission from Gilles, Leviton and Dooling Eds., copyright © 1983 The *Developing Human Brain*, Littleton, MA: John Wright.

distinguish "normal" from "abnormal" specimens, viewing all these pathological materials as abnormal to one degree or another. Second, they used a quantified scale of degree of myelination instead of qualitative description (0 = no myelin, 1 = microscopic only, 2 = just visible, 3 = intensity approaching mature myelin). Third, at every age they made assessments of a number of cases in identical fashion, and reported the results according to the statistical distribution of quantitative scale scores, thus incorporating the realities of individual variation into the basic presentation of the data. Finally, two observers systematically assessed each of 53 carefully defined sites in each specimen, yielding a total of nearly 70,000 scores for analysis.

Most recently, Brody et al. (1984) extended this painstaking method of assessment to the first 2 postnatal years. They studied 171 cases, a sample with no overlap with the Gilles et al. sample described above. Differences in method included the use of the luxol fast blue stain instead of a hematoxylin stain for visualization of myelin, and the use of standard neuropathological blocks taken from strategically chosen brain regions including a sample of the most important structures. [Gilles et al. (1983) apparently used some specimens cut in such blocks and some whole brain sections.] In other respects their method followed that of Gilles et al. This material is not yet fully reported, but it promises to greatly extend and refine the findings of Yakovlev and Lecours (1967) on the first 2 postnatal years. Fortunately, a preliminary account of their findings is available.

With the exception of Lecours (1975; Lecours et al. 1983), none of the modern investigators has had a direct interest in correlating myelination sequences with behavioral maturation, and it would be unfair to impute any such intention to them. Nevertheless, their work provides a basis for speculation of this kind. Their data, as well as that of the older investigators, will be used in the remainder of this study. As noted by Gilles et al. (1983), these studies demonstrate a substantial degree of agreement, certainly in the sequence of myelination and, to a lesser extent, in its precise timing, across studies done at different times, by different investigators, using different methods, and with different purposes.

Parallels in Behavioral and Neuroanatomical Development

Myelination and the Development of Behavior

Several investigators have suggested that specific events and sequences of myelination have specific behavioral consequences. For example, the system subserving detection of postural orientation and vestibular stimulation (Figure 1, line 3) is fully myelinated before birth. As Korner (1972) has noted, this may explain the unique effectiveness of rocking stimulation in quieting the newborn, as well as the apparent positive effect of upright posture on alertness at this age. A more mundane consequence of this state of myelination of the vestibular

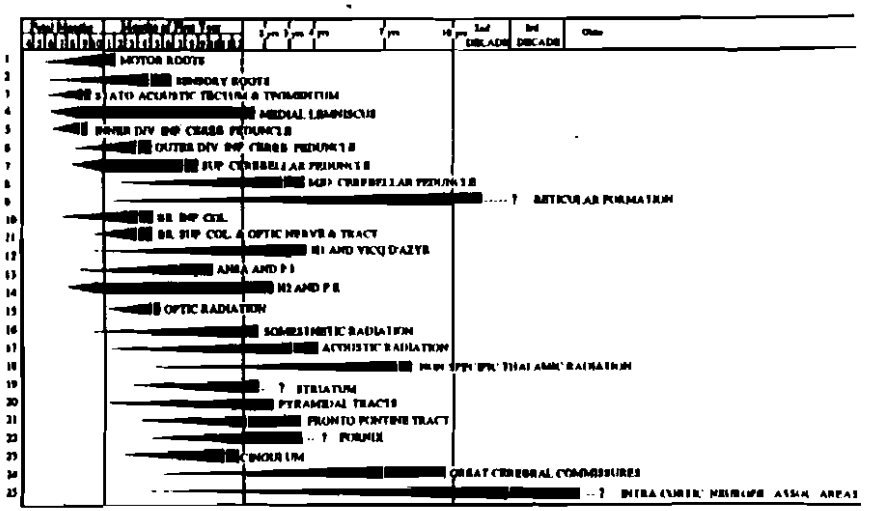

Figure 1. Cycles of myelination, adapted from Yakovlev and Lecours (1967). Widening of the bars indicates progressive intensity of staining and density of myelinated fibers. Interrupted bars to the right indicate approximate age range of termination of myelination process. From Yakovlev, P. I. and Lecours, A. R. "The myelogenetic cycles of regional maturation of the brain" in Minkowski, A. (Ed.) *Regional Development of the Brain in Early Life*, F. A. Davis Co., Phila., Pa., 1967, pp. 4–5. Copyright © Blackwell Scientific Publications, Ltd., Oxford.

system may be the concatenation of vestibular reflexes elicitable at birth (Peiper, 1963; Prechtl and Beintema, 1964; see next section for further discussion).

The major tracts of the visual system begin to show evidence of myelin staining just before birth and complete their myelination rapidly in the first few months of life (Figure 1, lines 11 and 15) (Brody et al., 1987; Yakovlev and Lecours, 1967). This corresponds to the rapid attainment of visual maturity in the same epoch. A detailed attempt to relate the maturation of visual perceptual capacity in early infancy to myelination sequences in the visual system constitutes one of the more convincing models of this kind in the literature (Bronson, 1974, 1982).

This pattern in the visual system contrasts markedly with the sequence of myelination of the auditory system, as noted by Lecours (1975; Lecours et al., 1983). The acoustic radiation to the cerebral cortex (Figure 1, line 17) has a very protracted course of myelination. It requires at least 1, possibly 2 years, corresponding to the pace of growth of the major function of the human auditory analyzer, namely language comprehension (Brody et al., 1987).

In general, the longer the axon, the more the cell gains by myelination. Thus, the myelination of the very long neurons of the pyramidal tracts (Figure 1, line 20) predicts quite well the dramatic gains in neuromuscular function during the

first year of life (Brody et al., 1987; Yakovlev and Lecours, 1967). A section through the spinal cord of a human newborn resembles sections seen in a patient with spinal transection (Rorke and Riggs, 1969). Further, the development of reflexive and sensorimotor behavior in the normal newborn resembles in several respects what would be expected in a neurological patient recovering from such an injury. These correspondences are discussed at some length below.

Finally, the great cortical association areas (Figure 1, line 25) may continue to gain myelin up to the age of 30 years. The increase in staining density in these areas is of a magnitude consistent with that of the known effects of experience on myelination (discussed previously) and may in fact represent such an effect, although the argument for a correlation is weakest with subtler degrees of change.

In a more sophisticated analysis of the myelination of the cortex, Gibson (1977, 1981) compared the myelination of afferents and efferents to different cortical layers in both rhesus monkeys and humans, in relation to the known facts about cognitive development in these two species. She noted that there is a reliable sequence of myelination among the six cortical layers in any given region of cortex, although the sequence occurs more rapidly in some areas than others. Specifically, just as the primary sensory and motor projection areas of cortex develop in advance of the association areas, the layers subserving communication with brainstem and spinal cord (I, IV, V, and VI) myelinate in advance of the layers subserving communication within the cortex itself (II and III). In particular, layers IV, V, and VI in the association cortex become myelinated between 15 and 24 months of age in humans, or later at a time when great advances in cognitive functioning, plausibly described as requiring intermodal integration of a high order, are taking place.

Other equally impressive correspondences between myelination sequences and behavioral development have received little or no attention in the literature. Some of the major tracts of the limbic system, which mediate the emotions, do not begin to myelinate until weeks or months after birth. The cingulum, linking the frontal lobe to the limbic system (Nauta, 1971), myelinates between 2 and 10 months (Figure 1, line 23); 4 months according to Brody et al., 1984). The fornix, a massive fiber bundle leaving the hippocampus, myelinates in the second half of the first year and later (Figure 1, line 22). Other major connection tracts of the limbic system myelinate in the first, second, third, and later years (Brody et al., 1987; Yakovlev and Lecours, 1967).

Finally, the corpus striatum and globus pallidus, as well as their fiber tracts, myelinate postnatally in the first and second years. These structures, long thought of as mere modulators of movement, are now known to participate intimately in the initiation of movement (Evarts, 1975). More interesting for present purposes, stimulation of sites within them has been shown to produce highly ritualized species-specific fixed action patterns that serve as social displays in squirrel monkeys (MacLean, 1978). This raises the possibility that if

there are any fixed action patterns in human social behavior, they may be controlled in part from homologous sites. None of these various tracts and structures is principally concerned with human information processing and problem solving with the possible exception of the fornix. All, however, are crucial to human social behavior, and all undergo dramatic changes during infancy.

The Neonatal Neurobehavioral Baseline

Neonatal neurological and behavioral assessment scales have produced a detailed characterization of human neurological status at birth. This characterization has served as the basis for analyses of the state of development of the neonatal nervous system (Minkowski, 1955; Peiper, 1963; Bronson, 1982). The peculiar concatenation of reflexes and movements, some essential for survival and continuous with mature functions (for example, the sucking and withdrawal reflexes), others precisely reminiscent of the signs of some neurological disorders and destined to wane with normal growth (such as athetoid movements and the plantar extensor reflex), has suggested to various observers that the neonate resembles a decorticate, decerebrate, midbrain, or even spinal organism in its level of function.

Prior to considering how best to model neonatal behavior in terms of neuroanatomical development, it is necessary to establish the fixed features of human neurobehavioral status at birth. This requires going beyond the European and American populations that have formed the basis of most generalizations. Occasional reports in the literature claim marked population-wide departures from what Europeans and Americans consider fundamental aspects of human neurological status at birth. Most notable among these was the report by Geber and Dean (1957) purporting to show that neonates of the Baganda population in Kampala, Uganda lacked certain reflexes considered obligatory in healthy European neonates: the automatic march reflex, the stepping reflex, the placing reflex, and the scarf sign. This lack was interpreted by the authors, and by many others who have cited their findings, as being indicative of precocity in African neonates. That is, they claimed that African neonates had already developed past the point at which these reflexes are elicitable, and they estimated the difference in developmental status to be on the order of 4 to 6 weeks.

Whatever the interpretation, this absence of major reflexes in a group of neonates would, if true, require rethinking of the status of the nervous system at birth. This finding, however, has not received confirmation from other studies. Warren (1972) reviewed the subject through the 1960s, and concluded that no such phenomenon had been shown in African neonates, citing three noncontirmatory studies and noting a number of defects in the original study. Super (1981) reviewed the subject more recently and concluded that "there is no reliable corroboration of Geber and Dean's claim of neurological precocity in African newborns. Substantial contradictory evidence now exists, as well as doubt about the original methodology" (p. 188).

This is not to say that there are no differences in the neonatal behavior and responsiveness of different racial groups. The possibility remains that the Moro reflex and the plantar extension reflex have higher thresholds and lower amplitudes in African than in European newborns. There is also some evidence of temperamental differences between Oriental and European newborns, with the Oriental infants showing less activity and less irritability (e.g., Freedman, 1974). Finally, there are numerous reports of statistically significant, but minor differences, in neonatal neurobehavioral status of different populations as measured by the Brazelton scale (Sameroff, 1979). These results, however, do not alter the general impression that a characteristic repertoire of reflexes and behavioral capabilities applies to the neonates of all human groups. (For detailed review see Super, 1981.)

Illustrative of the similarities are the results of the Prechtl neonatal neurological examination (discussed above) for a small sample of neonates in one African population, the !Kung San. Nine infants were examined between 8 and 12 days of age. During the examination, subtleties of threshold and amplitude of response were recorded, but for comparison purposes only the percentage of infants who unambiguously exhibited the reflex in response to the given stimulus, regardless of threshold amplitude, were used. Many of these responses are difficult to elicit in any neonate under the best of conditions. The proportions of infants responding among the !Kung San are within the range expected by chance for European and American samples. In particular, the reflexes reported by Geber to be absent in Baganda neonates after 4 days of age are present in !Kung neonates at 10 days of age.

Other investigators have obtained similar results within our African populations based on larger samples of neonates, and on samples representing several Asian populations, Australian aborigines, and New World Indians (Super, 1981). Thus, the general neurobehavioral capacity of human neonates appears adequately characterized by the normal expectable repertoire obtained through standard procedures for neurological examination in European and American populations (Prechtl and Beintema, 1964).

These behavioral and myelination data suggest that the neonate functions at a level consistent with full development of the peripheral nervous system, extensive development of the spinal cord and medulla, substantial development of the mesencephalon, and partial development of the prosencephalon, almost all below the cortical level.

Individual reflexes can be explained with reference to the state of development of specific structures (Minkowski, 1955; Peiper, 1963). For example, the tonic deviation of the head and eyes in the direction of body displacement, the doll's eye reflex, and the labyrinthine righting reflex owe their presence to the relative maturity of the vestibular portion of the eighth cranial nerve and its communications with the brainstem nuclei of the cranial nerves effecting the responses. The stepping and automatic march reflexes directly parallel similar reflexes in both

decorticate and decerebrate animal preparations, and are interpretable as reflecting the relatively advanced state of development of the spinal cord and medulla, with the exception of the descending cortical efferents, which provide inhibitory control. Finally, the best-studied neonatal reflex is the plantar extensor or Babinski reflex, which consists of a splaying outward and upward of the toes when the sole of the foot is stroked. This appears homologous to the same reflex in adult patients who have suffered damage to the corticospinal (pyramidal) tracts. In the neonate, this reflex almost certainly reflects the absence of myelin from the same tracts (Brain and Wilkinson, 1959; Ghez, 1981). Many similar arguments are possible with respect to sensory capacity at birth, for example, in the visual system (Bronson, 1974, 1982).

As noted by Bronson (1982), it is reasonable to think of the neonate as functionally subcortical. Although some potential for function exists in cortical systems, behavioral and physiological data suggest that this function is extremely rudimentry. An ingenious series of experiments, however, has shown that neonates are, to a limited extent, capable of intermodal transfer—exchange of information among different sensory modalities—a capability they were long thought to lack (Meltzoff and Moore, 1977; Meltzoff and Borton, 1979). Whether this reflects an unexpected level of functioning for unmyelinated intracortical association areas, or an unexpected role for subcortical structures in intermodal transfer, is impossible to determine at present (but see Gibson, 1981).

The Growth of Bipedal Locomotion: A Paradigmatic Case

Motor development sequences are largely genetically programmed. Research in the 1930s and 1940s by Gesell (Gesell and Amatruda, 1947), McGraw (1943), and others noted, and, in some instances, showed effects of experience, but such effects were invariably minor against the background of the temporal map of developmental milestones. McGraw explicitly embraced the notion of a species-specific neuromuscular development sequence and described it in detail for a longitudinally studied sample. Gesell and his colleagues made similar descriptions, supported by photographs, for a wider range of infant behavior patterns.

Shortly after his initial publications, Conel (1939–1967) began to rely on these two sets of behavioral descriptions in making functional interpretations of his large body of data on the regional maturation of the cerebral cortex. For example, he noted that by all measures area FA gamma of the precentral gyrus (the motor cortex), in the region of the hand, is, at 6 months of age, developmentally advanced compared to other areas of the motor strip. This corresponds well with behavioral evidence on the earlier maturation of hand control compared with control of other body parts. Another part of area FA gamma, the region of the lower extremities, is, in contrast, developmentally behind the rest of the motor cortex even at 15 months, corresponding to the continuing weakness of behavioral control of the lower limbs at that age.

To be sure, there are interesting variations in the timing of motor and sensorimotor milestones within and among populations (Super, 1981; Werner, 1972, 1979). At present it is not possible to partition such variation, whether within or between populations, into genetic, environmental, and genotype-environment interaction components. Nevertheless, the range of variation among individuals within samples considerably exceeds that among population means for the appearance of these milestones world-wide, despite much greater variation in environmental influences among populations. This consistency among population means suggests a species-specific and species-wide timing of events in motor and sensorimotor development. For example, independent sitting and visually directed reaching appear in the middle of the first year, independent rising to stand later that year, and independent walking and thumb-to-finger fine grasp early in the second year.

A useful paradigmatic case is provided by the emergence of bipedal locomotion, a species typical, centrally organized neuromotor action pattern shown by all normal adults—indeed by all normal 2 year olds—in our species. The mean age of attainment of one useful criterion, three steps taken without hands held, hovers around a year of age in many samples, usually falling between 11 and 14 months. Large samples studies in five European cities had means with 6 weeks of each other at the extremes (Hindley et al., 1966). Precocity for infants in developing countries, especially Africa, has frequently been claimed (Super, 1981; Werner, 1972, 1979). Some carefully designed and conducted studies, however, fail to show any difference, and one critical review of a large number of studies concluded that African infant precocity has not been demonstrated (Warren, 1972).

The claims for greatest precocity center on the early part of the first year of life. The means for African and other samples in developing countries for age at independent walking typically fall within the American and European range mentioned above. To take only one example, among the !Kung San, hunter-gathers of the Kalahari Desert in Botswana, deliberate efforts are made to accelerate the development of walking by means of seemingly appropriate tactics. These include extensively holding the infant in a sitting or standing posture long before independent maintenance of these postures is possible, exercising rudimentary walking capabilities, and so on. The curve for development of independent walking in these infants falls within the range of the corresponding curves for American infants (Konner, 1973, 1977).

Other cross-cultural variations in infant care that might be expected to alter the rate of development of this behavior also have little effect. The Hopi (Dennis, 1940; Dennis and Dennis, 1940) and Navajo Indians (Chisholm, 1983), for instance, traditionally restricted their infants much of the time by tightly swaddling them against cradleboards. This procedure did not substantially delay the age of first walking.

Finally, deliberate attempts to accelerate this maturational pattern experimentally under relatively controlled conditions usually met with little success (e.g., McGraw, 1935). One intervention study (Zelazo, et al., 1972) did succeed in producing an 8 week advancement of first independent walking by systematically exercising the neonatal automatic march reflex for the first 8 weeks of life. Although this suggests that extraordinary environmental modifications may alter the rate of development, more typical variations in rearing conditions do not have this effect.

In addition to regularities of developmental rate, a preponderance of evidence points to the existence of a species-typical developmental sequence. From the neonatal automatic march reflex, a centrally organized subcortical motor stereotype, to the mature gait of the 2 year old, with heel toe progression directly under the hips and synchronous alternate arm swinging, the developmental history of the bipedal locomotor pattern is for the most part characteristic and universal, and the timing of its major transition is narrowly defined.

Underlying this developmental history is a fairly clear plan of neural development. Classical studies linked the waning of the automatic march reflex as well as the ascendance of true walking to progressive development (including myelination, hypertrophy, and other growth changes) in the corticospinal tract from later prenatal life to the end of the second postnatal year (Minkowski, 1955; André Thomas, 1960). These changes result in increasing cortical inhibition of primitive spinal reflexes, stabilization of the postural stretch reflex, and voluntary control of coordinated limb movements. Accompanying the myelination of the corticospinal tracts are associated changes in the precentral (motor) gyrus, particularly the lower limb regions of the gyrus, over a similar developmental time course (Conel, 1939–1967). Functional evidence from clinical lesions shows that adults suffer profound loss of lower limb control in disease of the corticospinal tracts. Specifically, they exhibit the extensor plantar reflex of the neonate, known since Babinsky's 1896 description to be pathognomic of corticospinal tract conduction failure (see Ghez, 1981 for review). Thus, the development of improved function in this tract as a result of myelination is a likely candidate for a neural basis of the maturation of walking, and of the waning of the extensor plantar, or Babinsky reflex.

Modern concepts of the cortical and subcortical control of voluntary movement, however, have greatly expanded the range of neuroanatomical structures that may be involved in maturing locomotor skills. According to these concepts (Evarts, 1975; Allen and Tsukahara, 1974) both the basal ganglia and the cerebellum are implicated in goal corrected movement. Briefly, nonmotor cortex (i.e., the great majority of the cortex) initiates voluntary movement not primarily through a direct communication to the motor cortex (although such a connection may still play a role) but through transstriatopallidal and transcerebellar circuits. Nonmotor cortex projects to the basal ganglia and cerebellum, which project, via way-stations in the ventral anterior and ventral lateral thalamus, back to the motor and premotor cortex, respectively.

This widely accepted model must direct our attention to the myelination sequences of the major efferent pathways from the globus pallidus [the ansa lenticularis (Figure 1, line 13) and field H1 of Forel (line 12)], of the middle cerebellar peduncle (line 8), which carries cerebral cortical information to the cerebellum, and of the intrinsic fibers of basal ganglia (line 19). Collectively these structures exhibit a course of myelination that is highly consistent and as protracted as that of the corticospinal tract (line 20) and its somatosensory feedback pathway, the somesthetic radiation (line 16). This complex proposed circuitry of independent bipedal locomotion thus exhibits a cycle of myelination consistent with the use of the cycle to explain the maturation behavior.

Psychosocial Maturation during the First Year

Among many motoric, perceptual, and cognitive changes occurring during the first year of life are some transformations that are specifically social or emotional (Bowlby, 1969, 1973, 1980; Emde et al., 1976; Lewis and Rosenblum, 1978; Campos et al., 1983). Although all development is continuous, two critical transitions in infant psychosocial growth occur during the first year.

The first, achieved during the first 3 months of life, is characterized by a marked increase of social competence, from a neonatal level that is basically asocial or presocial, to a level of relatively advanced social expressiveness including mutual gaze interactions, contingent responsiveness in dyadic exchanges (Stern, 1974; Stern and Gibbon, 1977), and, most notably, the easily elicited social smile. Much data of a cross-cultural nature are available for one of these, the social smile (e.g., Gewirtz, 1965; Kilbride and Kilbride, 1975; Kilbride, 1980).

The second transition is slower, extending from about 5 to at least 15 months, with the most dramatic transformation taking place during the third quarter of the first year. It consists of the development of what is usually called attachment or attachment behaviors. One hallmark of this complex is crying or protest when the mother leaves the infant alone in a strange place, although numerous other specific behaviors with respect to the mother (or other primary caretaker) appear earlier, such as preferential greeting, approaching, and following. A related developmental phenomenon, the fear of strangers or stranger protest, also occurs during the second half of the first year.

The Emergence of Social Smiling. An early maturational development of a specifically social nature is the smile in response to a human face. In the human adult, smiling in greeting is universal, or at least exists universally as an option. It has been filmed and measured in the same form and context in societies on all continents, primitive and modern, some remote from the influence of the others (Eibl-Eibesfeldt, 1971a,b, 1983). Similarly, adults in widely distributed societies interpret pictures of smiles as signaling friendliness or happiness (Ekman, 1973). Young children exhibit the social smile in typical form and context and make the usual interpretation (Izard, 1977).

Quantitative variation in form and function of the smile may reflect the influence of learning, but such variation does not bear on the fundamental qualitative constancy of the behavior. It is as close as we are likely to come to a human fixed action pattern, or to a human species-specific social display. The smile evidently relates to the primate "playface," an open mouthed smile occurring during social play, and to the primate submissive closed mouth grin shown in greeting a dominant animal (Van Hooff, 1972; Andrew, 1963), but the relaxed friendly smile in social greeting is characteristically human (Blurton Jones, 1971).

For practical purposes, smiling is absent at birth and emerges during the first few months of postnatal life. Incidence of smiling in naturalistic social contexts or in experimental settings in which the infant is presented with a face is two orders of magnitude higher at 4 months of age than at term, and the response cannot be indisputably identified until some time in the second month (Ambrose, 1959; Emde, et al., 1976; Emde and Harmon, 1972; Spitz and Wolf, 1946; Sroufe and Waters, 1976). There is some variation in the early incidence and rate of emergence of smiling among samples in different environments. This variation is statistically significant but quantitatively minor (Gewirtz, 1965). Comparisons of infants among the Baganda of Uganda, the Samia of Kenya, and other cultures with American infants show little difference in the age of emergence of social smiling (Kilbride and Kilbride, 1975; Kilbride, 1980).

This growth process produces a marked change in parent–offspring relations. Mothers may report that they did not subjectively sense the existence of a relationship, or even that they did not love the infant, before the emergence of gaze fixation and competent social smiling (Robson, 1967; Robson and Moss, 1970). The absence of this care-eliciting behavior at birth is an evolutionary puzzle, the solution of which probably lies in the phylogenetic constraint on gestation length imposed by a narrowing birth canal on a slowing rate of growth (Konner, 1979, 1981).

A convincing developmental explanation of the emergence of social smiling has also eluded investigators. Well-formed nonsocial smiles occur regularly in neonates during rapid eye movement (REM) sleep and may be observed from 30 weeks of gestational age (Emde and Harmon, 1972; Emde et al., 1976; Wolff, 1963). Anencephalic infants with a mesencephalic level of functioning exhibit such smiles (Monnier, 1956), making telencephalic or diencephalic involvement in their regulation unlikely or at least not essential. Painstaking videotape studies of normal neonates show that highly coordinated and specific facial expressions, involving a number of sometimes widely separated elements of facial musculature acting in concert, are present at birth (Oster, 1978). Thus, at birth, with only lower brain functions mature, intricately timed facial muscle action patterns are already under complex central control. Also at birth, gaze fixation and even visual following of a face by the infant can be elicited, suggesting the existence of some underlying perceptual cognitive capacity (Als, 1977; Brazelton, 1973).

Since blind infants develop reliable social smiling only a month or two later

than sighted infants (Fraiberg, 1977; Freedman, 1964; see also Thompson, 1941), a crucial role for visual perception in the growth of the behavior can be ruled out. Since the mean age of the onset of social smiling in samples of low-risk mature infants can be better predicted from their postmenstrual than from their postnatal age (Brachfeld et al., 1980), a key role for associative or operant conditioning, both readily demonstrated later in infancy (Ahrens, 1954; Ambrose, 1959; Brackbill, 1958), also seems unlikely. Finally, since monozygotic twins are significantly more concordant in the rate of emergence of social smiling than dizygotic twins (Freeman, 1974), some genetic contribution to the individual variation is probable.

Thus, some central connection between the perceptual mechanism and the already well-formed motor output matures during the growth of this socially functional behavior. Some of the change may be perceptual and cognitive, rather than social or emotional. For example, by 2 months of age, visual following of eyes and faces substantially improves (Haith et al., 1977). By 3 months, an infant is most attracted to stimuli whose changes it can control ("contingently responsive stimuli" (Watson, 1972; Watson and Ramey, 1972)) or at least to stimuli (such as are provided by an indulgent caretaker) that change at a pace ideally suited to challenge infant attention (Stern, 1974; Stern and Gibbon, 1977) –faculties that are largely absent earlier. By 4 months, visual pattern memory emerges (Super et al., 1972). In addition to these essentially cognitive changes, some aspects of the maturing competence are undoubtedly specifically social, perhaps even in the ethological sense of the word, that is, "wired-in" for social functions.

Initial approaches to a developmental neurology of smiling must be indirect, but the following facts are noteworthy. Myelination of the motor roots of the fifth and seventh cranial nerves is completed prenatally (Langworthy, 1933; Rorke and Riggs, 1969). This finding is consistent with the mature form of nonsocial smiling even in premature neonates. The motor nuclei of these nerves are in close proximity to the pontine neurons believed to control REM sleep (McCarley and Hobson, 1975), which is the context in which these early nonsocial smiles most frequently occur.

In adults, voluntary and emotional control of the smile can each be lost separately as the result of regionally localized brain damage (Brain and Walton, 1969; Monrad-Krohn, 1924, 1927; Rinn, 1984). In facial paralysis from a corticospinal lesion above the level of the motor nuclei, voluntary retraction of the corners of the mouth is weak or absent, while smiling in appropriate emotional contexts is preserved. "Mimic" paralysis (the emotional form) is less clear in origin, but is believed to result from lesions of the basal ganglia (see Rinn, 1984 for review).

This notion receives strong support not only from stroke and injury studies but from the "masked face" syndrome of Parkinson's disease. This syndrome, which occurs early in the disease, consists of a marked facial unresponsiveness in the presence of emotion, but a preservation of voluntarily assumed facial expression. Since Parkinson's disease primarily affects the basal ganglia (or, more properly, the substantia nigra that projects to them), the masked face syndrome lends credence to the notion of regulation of social smiling from this part of the brain (Rinn, 1984).

In view of these findings—particularly if it is accepted that smiling is a species-typical social display—certain neuroethological concepts that would otherwise seem remote become relevant. MacLean and his colleagues have long maintained that the striatopallidal complex (the major part of the basal ganglia) plays a key role in the control of fixed social displays (MacLean 1978, 1985). They note the prominence of these structures (or their homologies) in birds and reptiles, taxa that (more exclusively than mammals) use fixed displays in their social behavior. More important, they have found and repeatedly confirmed that electrocoagulative lesions of the pars interna of the globus pallidus specifically abolish a species-typical fixed action pattern (genital presentation) that serves as a social display in the squirrel monkey, *Saimiri sciureus* (MacLean, 1978). No other deficits are observable in these monkeys, although these are assiduously sought, particularly in the realm of motor function, which is traditionally associated with striatopallidal circuits. This has led MacLean to redefine the striatopallidal complex as a regulator of species-specific displays, and to identify the globus pallidus as a particularly essential way-station for such displays.

From these findings, the following model of the growth of social smiling may be tentatively advanced. In late prenatal life the smile appears in mature form, during the high level of development of the fifth (trigeminal) and seventh (facial) nerves and their motor nuclei, but it does not appear in mature context. Its association with REM sleep perhaps bears some relationship to the relatively easy access of the pontine reticular formation to those motor nuclei.

In the course of the first few postnatal months, the response is brought into the realm of social control. Regional brain growth changes likely to be involved in this change are (1) sensory changes, perhaps, especially tectal (Figure 1, lines 10, 11, and 15); (2) motor changes, especially cerebellar (lines 5 and 6), and, most importantly, (3) changes in the striatopallidal complex, especially the globus pallidus and its efferents, and ansa lenticularis and the fields of Forel (lines 12–14), with the ansa and pars interna showing the most rapid change at this age (line 13).

The Growth of Social Fears. At the end of this phase transition, the social interaction is well established but relatively undiscriminating. It appears to the observer to be associated with positive emotion, but the emotion seems impersonal; almost anyone can elicit it and, despite subtle signs of discrimination of primary caretakers, strong emotional bonds do not appear to exist. This situation changes markedly in the second half-year. Strangers begin to be discriminated in social responding, often negatively, and increasingly so through the course of the

second 6 months (Morgan and Ricciuti, 1969; Tennes and Lampl, 1964; Lewis and Rosenblum, 1974); crying when left by the mother in a strange situation, with or without a strange person, becomes common, although it is certainly not universal (Bretherton and Ainsworth 1974; Ainsworth et al., 1978); vulnerability to the adverse effects of separations of substantial duration from primary caretakers becomes demonstrably more marked (Bowlby, 1973); and "attachment behaviors" such as following, clinging, and cuddling become frequent in relation to the primary caretaker(s), especially in strange situations or in the presence of strange persons (Ainsworth et al., 1978).

Such changes are, to be sure, not all functions of the growth of fear. They represent changes in the emotional valence of the interpersonal space of the infant that make certain key individuals very attractive while rendering the rest of the species less so, if not actually repelling. These changes are often characterized by primary caretakers as indicative of a deepening of the emotional bond they feel they share with the infant, and by theorists of affective development such as Bowlby (1969, 1980) and Ainsworth (Ainsworth, et al., 1978) as signaling the onset of the capacity for attachment, a major event in the growth of emotional and social competence.

Non-Western cultures exhibit similar behavioral patterns (Kagan, 1976; Super, 1981). Thus, the growth of social fears and the concomitant growth of attachment, as defined by these and related measures, appear to be universal features of the second half-year of human life (with much individual variation in the degree of overt expression). It is, at least in its ontogenetic timing, a species-specific feature of human behavioral organization. The percentage of infants who withdraw, fret, or cry when a stranger appears, who cry when left by the mother either alone or with a stranger, or who go to the mother rather than a stranger or a secondary caretaker when mildly apprehensive (Kagan et al., 1978) rises steadily from the middle of the first to the middle of the second year, whether the sample is drawn from the !Kung San of Botswana (who have 24-hour mother–infant physical contact in a dense social context), traditional Navajo Indians (who strap their infants into cradleboards much of the time), a remote Guatemalan Indian village (who have high mother–infant contact), an Israeli kibbutz (infant separated from the mother in a nursery except on afternoons and weekends), or various subcultures of the United States, including professional and working class socioeconomic levels (Konner, 1982; Chisholm, 1983; Kagan, 1976; Super, 1981). Among Chinese–American and Caucasian-American subcultures in Boston, infants who have 8 hours a day of day-care separating them from the mother do not differ significantly from control infants who have had no such separation on measures of social fear and attachment at any age, despite the fact that the day-care regime began before 4 months of age (Kagan et al., 1978). This latter finding is confirmed by similar studies of the effects of day-care on social behavior in other cities (Caldwell et al., 1970; Ricciuti, 1974; Brookhart and Hock, 1976; Blanchard and Main, 1979; Campos et al., 1983).

Similarly organized behavioral patterns, with species-typical motor components and ontogenetic timing, may be seen in the early postnatal development of higher primates (Blurton Jones, 1972; Rosenblum and Alpert, 1974) and other mammals (Scott, 1962). Analogous, although probably not homologous, events may be seen in the very early posthatching development of precocial birds (Sluckin, 1970). Observation in the environments of evolutionary adaptedness (Rheingold, 1963; Altman, 1979), including one such environment for humans (Konner, 1972, 1977, 1981), clearly suggests an adaptive role for such behaviors in two ways: prevention of predation and intergenerational transfer of adaptively relevant, acquired formation. The ontogenetic association of independent locomotion and active *imitation* with the growth of social fear and attachment behavior (in several species) supports, respectively, the two putative adaptive significances postulated for these infant emotions. The concatenation of fixed-action-pattern-like components with one another, and with apparently innate releasing mechanisms, in an organized, goal-corrected, predictable, "driven" fashion resembles, in important respects, other patterns that are usually called instincts.

Some evidence suggests that individual variation in the precise ontogenetic timing and in the degree of expression of the social fears is under genetic influence. In at least two studies, fear of strangers in infancy has been shown to have significantly higher concordance in identical than in fraternal twin pairs. One of these studies focused on the longitudinal pattern of growth of fear (Freedman, 1974), and the other on specific behaviors toward the stranger (Plomin and Rowe, 1978). No evidence exists for similar heritability of positive behavior toward caretakers, but little work has been done on this question. Possibly only the negative aspects of the fear/attachment complex have their individual variation under significant genetic influence.

For present purposes, however, it is the control not of individual variation but of universal features of maturation that is of interest. In view of the known facts relating fear and other strong emotions to the nuclei and pathways of the limbic system (see Isaacson, 1974, for review), it would be appropriate to examine this system for developmental events that might help to dispel the mystery of the rise of the social fears. In Papez's original formulation, the burden of the "stream of feeling" was laid for the first time on a core of circuitry, including especially the hippocampus, the fornix, the mammillary body, the mammillothalamic tract, the anterior nucleus of the thalamus, the cingulum bundle, and the cingulate cortex (Papez, 1937). It subsequently became clear that the main outgoing pathways of the amygdala—the ventral amygdalofugal path and the stria terminalis, various parts of the hypothalamus, and the septal area—also belong in the proposed emotional circuitry (MacLean, 1952; Nauta and Domesick, 1980).

In addition, Nauta delineated three major extensions of the system beyond these primarily diencephalic and older cortical nuclei and circuits: (1) a two-way communication with the frontal lobes as "the neocortex of the limbic system"

(Nauta, 1971); (2) direct fiber connections with the striatopallidal circuitry, via the ansa lenticularis and fields of Forel (Nauta and Mehler, 1966), perhaps providing a basis for species-specific displays of the emotions as proposed by MacLean (1978); and (3) various connections with the "limbic mid-brain," over the mammillary peduncle, the dorsal longitudinal fasciculus, the habenulointerpeduncular tract, and other pathways (Nauta, 1958; Nauta and Domesick, 1978), which probably constitute an important part of the nonendocrine effector output of the system, especially, but not exclusively, in the visceral realm. The connections of the limbic system and its relations with other brain circuitry have been authoritatively reviewed (Nauta and Domesick, 1980).

Turning to Figure 1, we note that at 3 months of age there is little or no myelin in the striatum, the fornix, and the cingulum (lines 19, 22, and 23), but that these have achieved almost the adult level of staining by the end of the first year (Yakovlev and Lecours, 1967). The mammillothalamic tract (bundle of Vic d'Azyr, line 12) also gains heavily in staining density during this period. Although there are other dramatic changes during the second half-year of life, they mainly relate to the neocortical and cerebellar control of movement (see above); as such they are likely to be less relevant to the growth of the emotions than are the structures just mentioned. With the absence of myelin on the fornix, the cingulum, and to a lesser extent the mammillothalamic tract, it is probable that the level of functioning in the Papez circuit is very poor at 3 months compared to its level of functioning at the end of the first year. Thus, it is not surprising to find that the emotional competence of the older infant is much greater, and it seems likely that more than an increase in information processing ability (an explanation offered by Kagan et al., 1978) is involved. Furthermore, the gains in myelin staining in the striatum and in the fiber fields of Forel (H1 and H2, lines 12 and 14) suggest the possibility that not only emotional competence, but also the ability to express emotion in motor action, are maturing at this age.

In addition, data from stimulation studies, lesion studies, and to a lesser extent, psychosurgical practice (for reviews see Gray, 1971; Valenstein, 1973; Isaacson, 1974) indicate the involvement of portions of the amygdala, the cingulum, the hypothalamus, and the limbic midbrain in the mediation of fear and anxiety. In the original classic experiments on the consequences of temporal lobe lesions, Klüver and Bucy found that removal of large portions of the temporal lobe in monkeys resulted in a syndrome including fearlessness, tameness, tendency to approach objects indiscriminately, mouthing of objects indiscriminately, and hypersexuality (Klüver and Bucy, 1937, 1939). With the exception of hypersexuality, which may require previously mature reproductive competence, the syndrome is in some respects reminiscent of behavior of normal human infants in the 4–5 month age range. Perhaps the absence of myelin in the limbic circuitry gives them a partial temporal lobe disconnection syndrome that mimics physiologically as well as phenomenologically the Klüver–Bucy syndrome in postoperative adult monkeys.

Whether or not these specific explanations relating infant emotionality to lesion studies are correct, no account of the striking changes in infant emotional competence during the second half-year of life will be satisfactory without an account of the relationship to the equally striking changes in limbic system structures know to underlie emotional behavior.

Discussion

This model is based on correlations, some of which are rather general in nature. Eventually, improvements in the methodology of relating myelination sequences to behavioral development will result from application of magnetic resonance imaging (MRI) to the maturing brain. MRI (also known as nuclear magnetic resonance, NMR) is demonstrably superior to computed tomography and other brain imaging methods, particularly in its ability to discriminate white from gray matter in the central nervous system, and is, for this reason, showing itself to be particularly useful in following demyelinating disorders such as multiple sclerosis. This imaging modality has the potential to produce quantified determinations of the degree of myelination in anatomically specific regions of the living, developing brain. Thus, the potential for correlations with developing behavior is extraordinary. Although strictly speaking these will still be mere correlations, they will be so much more precise and dynamic than the ones considered here as to constitute a completely new body of data against which to test these hypotheses. No matter how fine the correlations, however, such models will always be subject to criticism on the grounds that other structures that mature at the same time may be responsible for the behaviors in question.

One way to confirm the correlational models is to remove the pertinent developing structures and to observe the resulting behavioral deficits. These procedures generally rely on clinical materials that do not precisely parallel the desired lesion or on animal models that do not precisely parallel the function of the human brain. In one of the most impressive studies of this kind, Diamond (1985, and this volume) demonstrated a developmental sequence for performance on an object-retrieval task that matures during the second half of the first year in human infants. She attributed this development to maturation of the frontal cortex during the same time period, citing clinical and experimental lesion studies. She went on to show that rhesus monkeys exhibit the same sequence of development between 2 and 4 months of age and, most importantly, that the ability can be eliminated in adult rhesus monkeys by specific ablation of the dorsolateral prefrontal cortex, which is developing in both species at the ages at which they respectively develop the behavior (Diamond and Goldman-Rakic, 1983). This sequence of investigations provides a model for future research on the neuroanatomical bases of behavioral development. [Incidentally, the object retrieval task in question, which is, in effect, the object permanence task of

Piaget, has been shown to develop according to a pattern that deserves the status of a cross-cultural universal (Werner, 1979; Dasen and Heron, 1981).]

The model proposed in this chapter is also subject to criticism on the grounds that infant behavior has been properly studied in only a handful of the thousands of known human cultures. Although the cross-cultural studies reviewed are not exhaustive, they do sample a widely representative selection of human populations around the world. These populations are mutually independent. Some are essentially independent of Western influence. Most are very different from one another in culture, ecology, nutrition, child training, and other characteristics of the environment that might jeopardize the hypothesis of universality. These systematic studies of a few populations draw support from descriptive and anecdotal materials from a much larger range of cultures. At this point, the burden of proof rests on whose who assert the possibility of a marked cultural departure in infant behavior or developmental sequence. Fortunately, the field of cross-cultural psychology is developing rapidly, and will in the near future settle the question for many behaviors (Triandis and Lambert, 1980–1981).

In the model of social development proposed here, an essentially presocial phase during the neonatal period is followed within 3 months by a highly, but indiscriminately, social phase of which the hallmarks are effective sustained gaze contact and reliable social smiling. This, in turn, is followed, by the last quarter of the first year, by a type of sociability the very essence of which is discrimination. It is characterized by preferential direction of positive social behavior toward one of a few individuals with an absence of response toward others and frequently negative responses toward strangers. This is usually called the attachment phase of infant social development. Finally, the maturation of higher cognitive functions, particularly, but not exclusively, in the realm of language, serves by around age 3 years to begin to terminate the intense phase of attachment to the primary caretaker and to deliver the child into a wider social and cultural world (Lenneberg, 1967; Lecours, 1975; Lecours et al., 1983).

It is interesting to note some parallels between this sequence of three phases of social development and the three phases in the evolutionary sequence postulated by MacLean (1973, 1995). Briefly, MacLean has argued that the human brain consists of at least three levels of systemic functioning that, while certainly not separate in their ongoing activity, are usefully separated for heuristic purposes. Each represents the contribution made to brain structure by a major epoch of phylogeny: the reptilian brain, or "R-complex," roughly corresponding to the level of the basal ganglia in humans; the paleomammalian brain, representing the major neuroanatomical advance of the early mammals over the reptiles, and corresponding to the level of the limbic system in humans; and the neomammalian brain, corresponding to the level of the neocortex, which has increased steadily, especially in certain lines, throughout mammalian evolution.

The concept of recapitulation of phylogeny by ontogeny has been frequently criticized, and at one time was certainly exaggerated by evolutionary biologists, but it may remain valid to a limited extent (Gould, 1977; Konner, 1981; Gibson, 1981, 1983; Parker and Gibson, 1979). As applied to the proposed sequence of psychosocial development, it is reasonable to hypothesize the following. The human neonate has a severely restricted social repertoire, consisting mainly of expressions of distress that are probably not, strictly speaking, social. Within a few months the infant is capable of engaging adults in compelling social interactions, utilizing "wired-in" ritualized motor patterns, notably the social smile. Smiling as a social display may be functionally similar to the social display behaviors of other animals, including reptiles, which MacLean has shown to be mediated by the basal ganglia (MacLean, 1978, 1985), just as emotional smiling appears to be in humans (Monrad-Krohn, 1924, 1927; Rinn, 1984).

By the end of the first years, at the latest, social behavior has taken on a much more complex emotional depth, including such phenomena as attachment, fear of separation, and fear of strangers. This developmental sequence depends, in large part, on the maturation of the major fiber tracts of the limbic system, and on the development of their nuclei of origin and destination. This corresponds closely to the evolutionary advance represented by the paleomammalian brain in MacLean's model, both at neuroanatomical and behavioral levels. In fact, what MacLean views as the crucial functions of the paleomammalian brain are exemplified best by the very social capacities, summarized by a term attachment, that emerge in the human infant at this time (MacLean, 1985).

Finally, the maturation of higher neocortical and cognitive functions (Gibson, 1981; Parker and Gibson, 1979), ultimately including language (Lecours, 1975; Lecours et al., 1983), provides a reasonable correspondence with the phylogenetic emergence of the neocortex, MacLean's neomammalian brain, superimposing on the two older systems a series of new neurobehavioral functions, but without eliminating or suppressing completely the older functions of the other systems. Thus, the model proposed here, although conceived independently of MacLean's "triune brain" model, appears to have something in common with it as a recapitulation, in part, of the proposed events of phylogeny.

Finally, from a psychiatric viewpoint, it is interesting to note that this model in a sense attempts what Freud tried unsuccessfully to do at a much earlier stage in the history of neuroscience—to provide an account of the fixed features of neurological development that could be used as a basis for the interpretation of psychosocial development. Freud's theory of libidinal energy, which he postulated as migrating from one part of the central nervous system to another according to an orderly development plan (Freud, 1905), can, it is to be hoped, soon be replaced by an account of the actual features of nervous system development, as a basis for the analysis of psychosocial growth.

Summary and Conclusions

A number of universals of behavior and developmental sequence in infancy exist that appear to be independent of variations in cultural and environmental context within the normal human range. These universals may relate the underly-

ing species-typical sequences of regional maturation of the brain. Myelination sequences, in particular, serve as a useful index of regional brain maturation and, with some reservations, are an important basis for developing function. The neurobehavioral status of the neonate is subject to modeling with reference to the degree of myelination and other aspects of neural developmental status at birth. In the realm of motor development, particularly with reference to the maturation of bipedal walking, enough is known to give a fairly convincing explanation by relying on myelination sequences in the corticospinal tract, the postthalamic somesthetic radiation, and selected features of the extrapyramidal and cerebellar systems. Finally, in the realm of psychosocial development, it is possible to propose a tentative explanation of the two main transitions of the first year of life, the emergence of social smiling and the emergence of attachment, by reference to developmental events indicated by the myelination of the circuitry of the basal ganglia and of the limbic system, respectively. Further research can be expected to expand and improve these models by providing a broad-based assessment of patterns of regional brain maturation and by further elucidating, through clinical and experimental studies, the neuroanatomical bases of complex behavior.

This model of the development of social behavior in infancy is related to the evolutionary model proposed by MacLean (1973, 1985) and commonly known as "the triune brain." Interesting parallels are drawn between the major events of psychosocial development in infancy and the phylogenetic sequence of MacLean, with the suggestion that ontogeny in this area of development recapitulates, to a limited extent, phylogeny. The model also has the interesting property of serving the function that was meant to be served by Freud's theory of libidinal development, but using known facts of nervous system development instead of dubious and speculative theory.

It is concluded that the strategy proposed has been successfully pursued, and that the model outlined has conceptual and heuristic value. Further research will undoubtedly improve the model by broadening the base for generalization about regional brain maturation and by elucidating the neuroanatomical bases of complex behavior.

Acknowledgments

The author thanks B. A. Brody, I. Devore, K. Gibson, J. Kagan, A. R. Lecours, W. Nauta, C. Super, and the late P. I. Yakovlev for valuable discussions. B. A. Brody generously provided access to unpublished data from her study of postnatal myelination. P. I. Yakovlev, with his characteristic gracious generosity, acted as a guide through the great maze of the Yakovlev Collection during four visits and facilitated the interpretation and photography of specimens relevant to normal postnatal myelination. Mohamed Haleem, Curator of the Collection, was also of great help. The late Norman Geschwind commented helpfully in writing on the proposal for this review. Earlier work on the !Kung San frequently referred to here was made possible by the help of I. DeVore, N. Howell, R. B. Lee, and M. Shostak, and by the support of the National Science Foundation, the National Institute of Mental Health, the Harry Frank Guggenheim Foundation, the Spencer Fund, and the Milton Fund. Earlier work leading more directly to this review was supported by the Social Science Research Council and the John Simon Guggenheim Memorial Foundation.

Notes

1. This uniformity also applies to several hundred other brains in the Yakovlev collection with specific neuropathological abnormalities.
2. The method is extremely expensive and considered prohibitive for most purposes today. Thus the Yakovlev Collection, located at the Armed Forces Institute of Pathology in Washington, D.C., remains a major resource for studies requiring whole brain reconstruction, whether for tracking of long fiber tracts, supplying the larger context of a focus of interest, or other reasons. Many further studies on this same material are possible. An up-to-date and independent reappraisal of the method and the current status and potential of the Collection is given by Kretschmann et al. (1979).

References

Adams, R. D., and Victor, M. (1981). *Principles of neurology, 2nd ed.* New York: McGraw Hill.

Ahrens, R. (1954). Beitrag zur Entwicklung des Physiognomie und Mimmerkennens. *Zeitschrift für experimentell und angewandte Psychologie, II*(3), 412–454 and II(4), 599–633.

Ainsworth, M. D. S., Blehar, M. C., Waters, E., and Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation.* Hillsdale N.J.: Lawrence Erlbaum.

Allen, G. I., and Tsukahara, N. (1974). Cerebrocerebellar communication systems. *Physiological Review, 54*, 957–1006.

Als, H. (1977). The newborn communicates. *Journal of Communication, 27*(2), 66–73.

Altman, J. (1979). *Baboon mothers and infants.* Cambridge, MA: Harvard University Press.

Ambrose, J. A. (1959). The development of the smiling response in early infancy. In B. M. Foss (Ed.), *Determinants of infant behavior I.* London: Methuen.

André-Thomas, Chesni, Y., and St. Anne Dargassies, A. (1960). *The neurological examination of the infant. Little Club Clinics in Developmental Medicine, No. 1.* London: Heinemann.

Andrew, R. J. (1963). Evolution of facial expressions. *Science, 142*, 3595, 1034–1041.

Angulo Y Gonzalez, A. W. (1929). Is myelinogeny an absolute index of behavioral capability? *Journal of Comparative Neurology, 48* 459–464.

Bayley, N. (1965). Comparisons of mental and motor test scores for ages 1–15 months by

sex, birth order, race, geographical location and education of parents. *Child Development, 36*, 379–411.

Bayley, N. (1969). *Manual for the Bayley Scales of Infant Development*. New York: The Psychological Corporation.

Beintema, D. J. (1968). *A neurological study of newborn infants. Clinics in Developmental Medicine No. 28*. London: Heinemann.

Blanchard, M., and Main, M. (1979). Avoidance of the attachment figure and socioemotional adjustment in day-care infants. *Developmental Psychology, 4*, 445–446.

Blinkov, S. M., and Glezer, I. I. (1968). *The human brain in figures and tables: A quantitative handbook*. New York: Basic/Plenum.

Blurton Jones, N. G. (1971). Criteria for use in describing facial expressions. *Human Biology, 43*(3), 365–413.

Blurton Jones, N. G. (1972). Comparative aspects of mother-child contact. In N. G. Blurton Jones (Ed.), *Ethological studies of child behavior*. New York: Cambridge University Press.

Bostock, H., Hall, S. M., and Smith, K. H. (1981). Demyelinated axons form "nodes" prior to remyelination. *Journal of Physiology (London), 308*, 21P.

Bowlby, J. (1969). *Attachment*. New York: Basic Books.

Bowlby, J. (1973). *Separation: Anxiety and anger*. New York: Basic Books.

Bowlby, J. (1980). *Loss: Sadness and depression*. New York: Basic Books.

Brackbill, Y. (1958). Extinction of the smiling response in infants as a function of reinforcement schedule. *Child Development, 29*, 114–124.

Brachfeld, S., Goldberg, S., and Sloman, J. (1980). Parent infant interaction in free play at eight and twelve months: Effects of prematurity and immaturity. *Infant Behavior and Development, 3*, 289–305.

Brain, L., and Walton, J. N. (1969). *Brain's diseases of the nervous system*. Oxford: Oxford University Press.

Brain, R., and Wilkinson, M. (1959). Observations on the extensor plantar reflex and its relationship to the functions of the pyramidal tract. *Brain, 82*, 297–320.

Brazelton, T. B. (1973). *Neonatal behavioral assessment scale. Clinics in Developmental Medicine, No. 50*. London: Heinemann.

Bretherton, I., and Ainsworth, M. (1974). Response of one-year-olds to a stranger in a strange situation. In M. Lewis and L. Rosenblum (Eds.), *The origins of fear*. New York: Wiley.

Brody, B. A., Kinney, H. C., Kloman, A. S., and Gilles, F. H. (1984). Sequences of human postnatal myelination. Paper presented at the American Association of Neuropathologists, 60th Annual Meeting, November.

Brody, B. A., Kinney, H. C., Kloman, A. S., and Gilles, F. H. (1987). Sequence of central nervous system myelination in human infancy. I. An autopsy study of myelination. *Journal of Neuropathology and Experimental Neurology, 46*, 283–301.

Bronson, G. W. (1974). The postnatal growth of visual capacity. *Child Development, 45*, 873–890.

Bronson, G. W. (1982). Structure, status and characteristics of the nervous system at birth. In P. Stratton (Ed.), *Psychobiology of the human newborn*. New York: Wiley.

Brookhart, J., and Hock, E. (1976). The effects of experimental context and experimental

background on infant's behavior toward the mother and a stranger. *Child Development, 47*, 333–340.

Cairns, R. B. (Ed.). (1974). *Social interaction: Methods, analysis and illustration*. New York: Lawrence Erlbaum.

Caldwell, B. M., Wright, C., Honig, A., and Tannenbaum, J. (1970). Infant day-care and attachment. *American Journal of Orthopsychiatry, 40*, 397–412.

Campos, J. J., Barrett, K. C., Lamb, M. E., Goldsmith, H. H., and Stenberg, C. (1983). Socioemotional development. In P. H. Mussen (Ed.), *Handbook of child psychology*, 4th ed., Vol. 2: *Infancy and developmental psychobiology* (pp. 783–916). New York: Wiley.

Carnow, T. B., Carson, J. H., Brostoff, S. W., and Hogan, E. L. (1984). Myelin basic protein gene expression in quaking, jimpy, and myelin synthesis-deficient mice. *Developmental Biology, 106*, 38–44.

Carter, S., and Gold, A. P. (1974). *Neurology of infancy and childhood*. New York: Appleton-Century-Crofts.

Chisholm, J. (1983). *Navajo infancy: An ethological study of child development*. New York: Aldine.

Conel, J. (1939–1967). *The postnatal development of the human cerebral cortex*, Vols. I–VIII. Cambridge, MA: Harvard University Press.

Dasen, P. R., and Heron, A. (1981). Cross-cultural tests of Piaget's theory. In H. C. Triandis and A. Heron (Eds.), *Handbook of cross-cultural psychology*, Vol. IV: *Developmental psychology* (pp. 295–341). Boston: Allyn and Bacon.

Dennis, W. (1940). Does culture appreciably affect patterns of infant behavior? *Journal of Social Psychology, 12*, 305–317.

Dennis, W., and Dennis, M. G. (1940). The effect of cradling practices upon the onset of walking in Hopi children. *The Pedagogical Seminary and Journal of Genetic Psychology, 56*, 77–86.

Diamond, A. (1985). Development of the ability to use recall to guide action, as indicated by infants' performance on $A\bar{B}$. *Child Development, 56*, 868–883.

Diamond, A., and Goldman-Rakic, P. (1983). Comparison of performance on a Piagetian object permanence task in human infants and rhesus monkeys: Evidence for involvement of prefrontal cortex. *Neuroscience Abstracts* (Pt. 1), *9*, 641.

Diamond, M. C., Krech, D., and Rosensweig, M. R. (1964). The effects of an enriched environment on the histology of the rat cerebral cortex. *Journal of Comparative Neurology, 123*, 111–120.

Diamond, M. C., Johnson, R. E., Protti, A. M., Ott, C., and Kajisa, L. (1985). Plasticity in the 904-day-old male rat cerebral cortex. *Experimental Neurology, 87*, 309–317.

Eibl-Eibesfeldt, I. (1971a). Vorprogrammierung in menschlichen Sozialverhalten. *Mitteilungen an die Max-Planck-Gesellschaft, 5*, 307–338.

Eibl-Eibesfeldt, I. (1971b). Zur Ethologie menschlichen Grussverhaltens: II. Das Grussverhalten und einige andere Muster freundlicher Kontaktaufnahme der Waika Indianer (Yanoama). *Zeitschrift für Tierpsychologie, 29*, 196–213.

Eibl-Eibesfeldt, I. (1983). Patterns of parent-child interaction in a cross-cultural perspective. In A. Oliverio and M. Zappella (Eds.), *The behavior of human infants*. New York: Plenum.

Ekman, P. (1973). Cross-cultural studies of facial expression. In P. Ekman (Ed.), *Darwin and facial expression*. New York: Academic Press.

Emde, R. N., and Harmon, R. J. (1972). Endogenous and exogenous smiling system in early infancy. *Journal of the American Academy of Child Psychiatry, 11*(2), 177–200.

Emde, R. N., Gaensbauer, T. J., and Harmon, R. J. (1976). Emotional expression in infancy: A biobehavioral study: *Psychological Issues Monograph*, 10(37) 1–192.

Evarts, E. B. (1975). The third Stevenson lecture. Changing concepts of central control of movement. *Canadian Journal of Physiology, 53*, 191–200.

Field, T. M., Sostek, A. M., Vietze, P., and Leiderman, P. H. (Eds.). (1981). *Culture and early interactions*. Hillsdale, N.J.: Lawrence Erlbaum Associates.

Flechsig, P. (1920). *Anatomie des Menschlichen Gehirns und Rückenmarks auf myelogenetischer Grundlage*. Leipzig: Georg Thieme.

Fraiberg, S. (1977). *Insights from the blind: Developmental studies of blind children*. New York: Basic Books.

Freedman, D. G. (1964). Smiling in blind infants and the issue of innate vs. acquired. *Journal of Psychology and Psychiatry, 5*, 171–184.

Freedman, D. G. (1974). *Human infancy: An evolutionary perspective*. New York: Wiley.

Freeman, B. (1978). Myelin sheath thickness and conduction latency groups in the cat optic nerve. *Journal of Comparative Neurology, 181*, 183–196.

Freud, S. (1905). *Three essays on the theory of sexuality*. Standard edition of the complete psychological works of Sigmund Freud, (Vol. 7, pp. 135–245). London: Hogarth.

Friede, R., and Samorajski, T. (1967). The relation between the number of myelin lamellae and the axon circumference in fibres of vagus and sciatic nerves of mice. *Journal of Comparative Neurology, 130*, 223–232.

Geber, M., and Dean, R. F. A. (1957). The state of development of new born African children. *Lancet, 1*, 1216–1219.

Geschwind, N., and Galaburda, A. (1985). Cerebral lateralization and cortical dominance. Parts I, II and II. *Archives of Neurology, 42*(5, 6, and 7).

Gesell, A., and Amatruda, C. S. (1947). *Developmental diagnosis* 2nd ed. New York: Hoeber.

Gewirtz, J. L. (1965). The course of infant smiling in four child-rearing environments in Israel. In B. M. Foss (Ed.), *Determinants of infant behavior III* (p. 161–180). London. Metheun; New York: Wiley.

Ghez, C. (1981). Cortical control of voluntary movement. In E. R. Kandel and J. H. Schwartz (Eds.), *Principles of neural science* (pp. 323–333). New York: Elsevier.

Gibson, K. R. (1977). Brain structure and intelligence in macaques and human infants from a Piagetian perspective. In S. Chevalier Skolnikoff and F. E. Poirer (Eds.) *Primate biosocial development* (pp. 113–157). New York: Garland.

Gibson, K. R. (1981). Comparative neuro-ontogeny: Its implications for the development of human intelligence. In G. Butterworth (Ed.), *Infancy and epistemology* (pp. 52–82). Brighton, England: Harvester.

Gibson, K. R. (1983). Comparative neurobehavioral ontogeny: the constructionist perspective in the evolution of language, object manipulation and the brain. In E. DeGrolier (Ed.), *Glossogenetics* (pp. 41–46). New York: Harwood Academic Press.

Gilles, F. H., Leviton, A., and Dooling, E. C. (1983). *The developing human brain: Growth and epidemiologic neuropathology*. Boston: John Wright/PSG.

Globus, A., Rosensweig, M. R., Bennett, E., and Diamond, M. C. (1973). Effects of differential experience on dendritic spine counts. *Journal of Comparative and Physiological Psychology, 82*, 175–181.

Gould, S. J., (1977). *Ontogeny and phylogeny*. Cambridge, MA: Harvard University Press.

Gray, J. A. (1971). *The psychology of fear and stress*. New York. McGraw Hill.

Griffiths, R. (1954). *The abilities of babies*. London: University of London Press.

Gyllensten, L., and Malmfors, T. (1963). Myelination of the optic nerve and dependence on visual function. *Journal of Embryology and Experimental Morphology, 11*, 255–266.

Haith, M. M., Bergman, T., and Moore, M. J. (1977). Eye contact and face scanning in early infancy. *Science, 198*, 853–855.

Heilman, K. M., and Valenstein, E. (Eds.). (1979). *Clinical neuropsychology*. New York: Oxford.

Hindley, C. B., Filliozat, A. M., Klackenberg, G., Nicolet-Meister, D., and Sand, E. A. (1966). Differences in the age of walking in five European longitudinal samples. *Human Biology, 38*, 364–379.

Hogan, E. L., and Greenfield, S. (1984). Animal models of genetic disorders of myelin. In P. Morell (Ed.), *Myelin*, 2nd ed. (pp. 489–534). New York: Plenum.

Huttenlocher, P. R. (1970). Myelination and the development of function in immature pyramidal tract. *Experimental Neurology, 29*, 405–415.

Huxley, A. F., and Stämpfli, R. (1949). Evidence for saltatory conduction in peripheral myelinated nerve fibres. *Journal of Physiology, 108*, 315–339.

Isaacson, R. L. (1974). *The limbic system*. New York. Plenum Press.

Izard, C. E. (1977). *Human Emotions*. New York: Plenum Press.

Jacobson, M. (1978). *Developmental neurobiology*. New York: Plenum Press.

Kagan, J. (1976). Emergent themes in human development. *American Scientist, 64*(2), 186–196.

Kagan, J. (1984). *The natural child*. New York: Basic Books.

Kagan, J., Kearsley, R., and Zelazo, P. (1978). *Infancy: Its place in human development*. Cambridge, MA: Harvard University Press.

Keene, M. F. L., and Hewer, E. E. (1931). Some observations on myelination in the human central nervous system. *Journal of Anatomy, 66*, 1–13.

Kilbride, J. E., and Kilbride, P. L. (1975). Sitting and smiling behavior of Baganda infants. The influence of culturally constituted experience. *Journal of Cross Cultural Psychology, 6*, 88–107.

Kilbride, P. L. (1980). Sensorimotor behavior of Baganda and Samia infants: A controlled comparison. *Journal of Cross-Cultural Psychology, 11*, 131–152.

Kingsley, J. R., Collins, G. H., and Converse, W. K. (1970). Effect of sciatic neurectomy on myelinogenesis in the rat spinal cord. *Experimental Neurology, 26*, 498–508.

Kinsbourne, M., and Hiscock, M. (1983). The normal and deviant development of functional lateralization of the brain. In P. Mussen (Ed.), *Handbook of child psychology*, 4th ed., Vol. II. New York: Wiley.

Klaus, M., and Kennell, J. (1976). *Maternal infant bonding*. St. Louis: Mosby.

Klüver, H., and Bucy, P. C. (1937). "Psychic blindness" and other symptoms following bilateral temporal lobectomy in Rhesus monkeys. *American Journal of Physiology, 119*, 352–353.

Klüver, H., and Bucy, P. C. (1939). Preliminary analysis of functions of the temporal lobes in monkeys. *Archives of Neurological Psychiatry, 42*, 979–1000.

Knobloch, H. (1958). Precocity of African children. *Pediatrics, 22*, 601–604.

Konner, M. J. (1972). Aspects of the developmental ethology of a foraging people. In N. G. Blurton Jones (Ed.), *Ethological studies of child behavior*. Cambridge: Cambridge University Press.

Konner, M. J. (1973). Newborn walking: Additional data. *Science, 179*, 307.

Konner, M. J. (1974). Relations among infants and juveniles in comparative perspective. In M. Lewis and L. Rosenblum (Eds.), *Friendship and peer relations*. New York: Wiley. (Reprinted in *Social Science Information*, 1976, *15*(2), 371–402.)

Konner, M. J. (1976). Maternal care, infant behavior and development among the !Kung. In R. B. Lee and I. Devore (Eds.), *Kalahari hunter-gatherers*. Cambridge, MA: Harvard University Press.

Konner, M. J. (1977). Infancy among the Kalahari Desert San. In H. Liedermann, S. Tulkin, and A. Rosenfeld (Eds.), *Culture and infancy*. New York: Academic Press.

Konner, M. J. (1979). Biological bases of social development. In M. W. Kent and J. E. Rolf (Eds.), *Primary prevention of psychopathology*. Vol II: *Social competance in children*. Hanover: New England University Press.

Konner, M. J. (1981). Evolution of human behavior development. In R. H. Munroe and B. B. Whiting (Eds.), *Handbook of cross-cultural development*. New York: Garland.

Konner, M. M., and Worthman, C. (1980). Nursing frequency, gonadal function and birth spacing among !Kung hunter-gatherers. *Science, 207*, 788–791.

Korner, A. (1972). State as variable, as obstacle and as mediator of stimulation in infant research. *Merrill-Palmer Quarterly, 18*, 77–94.

Kretschmann, H. J., Schleicher, A., Grottschreiber, J. F., and Kullmann, W. (1979). The Yakovlev Collection: A pilot study of its suitability for the morphometric documentation of the human brain. *Journal of the Neurological Sciences, 43*, 111–126.

Langworthy, O. (1933). Development of behavior patterns and myelination of the nervous system in the human fetus and infant. *Contributions to Embryology, 139*, 1–57.

Lecours, A. R. (1975). Myelogenetic correlates of the development of speech and language. In E. H. Lenneberg and E. Lenneberg (Eds.), *Foundations of language development*, (Vol. 2, pp. 75–94). New York: Academic Press.

Lecours, A. R., Lhermitte, F., and Bryans, B. (Eds.). (1983). *Aphasiology*. London: Balliere Tindall.

Lenneberg, E. H. (1967). *Biological foundations of language*. New York: Wiley.

LeVay, S., Wiesel, T. N., and Hubel, D. H. (1980). The development of ocular dominance columns in normal and visually deprived monkeys. *Journal of Comparative Neurology, 191*, 1–51.

Lewis, M., and Rosenblum, L. A. (Eds.). (1974). *The origins of fear*. New York: Wiley.

Lewis, M., and Rosenblum, L. A. (Eds.). (1978). *The development of affect*. New York: Plenum.

MacLean, P. D. (1952). Some psychiatric implications of physiological studies on

frontotemporal portion of limbic system (visceral brain). *Electroencephalography and Clinical Neurophysiology, 4*, 407–418.

MacLean, P. D. (1973). *A triune concept of brain and behavior. The Hincks Memorial Lectures*. Toronto: University of Toronto.

MacLean, P. D. (1978). Effects of lesions of globus pallidus on species-typical display behavior of squirrel monkeys. *Brain Research, 149*, 175–196.

MacLean, P. D. (1985). Brain evolution relating to family, play and the separation call. *Archives of General Psychiatry, 42*, 405–417.

Martinez, A. J., and Friede, R. L. (1970). Changes in nerve cell bodies during the myelination of their axons. *Journal of Comparative Neurology, 138*, 329–338.

Matthews, M. A. (1968). An electron microscopic study of the relationship between axon diameter and the initiation of myelin production in the peripheral nervous system. *Anatomical Record, 161*, 337–352.

McCarley, R. W., and Hobson, J. A. (1975). Discharge patterns of cat brain stem neurons during desynchronized sleep. *Journal of Neurophysiology, 38*, 751–766.

McDonald, W. I., and Sears, T. A. (1970). The effects of experimental demyelination on conduction in the central nervous system. *Brain, 93*, 583–598.

McGraw, M. (1935). *Growth: A study of Johnny and Jimmy*. New York: Appleton-Century.

McGraw, M. (1943). *The neuromuscular maturation of the human infant*. New York: Columbia University Press.

Meltzoff, A., and Borton, R. W. (1979). Intermodal matching by human neonates. *Nature (London), 282*, 403–404.

Meltzoff, A., and Moore, M. K. (1977). Imitation of facial and manual gestures by human neonates. *Science, 198*, 75–78.

Minkowski, M. (1955). Quelques réflexions sur la neurophysiologie du nouveau né et du nourrisson et ses relations avec celle du foetus. *Revue Neurologique, 93*, 241–256.

Monnier, M. (1956). The behaviour of newborn anencephalics with varying degrees of anencephaly. In J. M. Tanner and B. Inhelder (Eds.), *Discussion on child development* (Vol. 2, pp. 237–239). London: World Health Organization/Tavistock.

Monrad-Krohn, G. H. (1924). On the dissociation of voluntary and emotional innervation in facial paralysis of central origin. *Brain, 47*, 22–35.

Monrad-Krohn, G. H. (1927). A few remarks on the question of dissociation between voluntary and emotional innervation in peripheral facial paralysis. *Acta Psychiatrica et Neurologica, 3*, 35–39.

Moore, C. L., Kalil, R., and Richards, W. (1976). Development of myelination in optic tract of the cat. *Journal of Comparative Neurology, 165*, 125–136.

Morell, P. (Ed.). (1984). *Myelin* 2nd ed. New York: Plenum.

Morgan, G. A., and Ricciuti, H. N. (1969). Infants responses to strangers during the first year. In B. M. Foss (Ed.), *Determinants of infant behavior* (Vol. 4, pp. 253–272). London: Methuen.

Mussen, P. H. (Ed.). (1983). *Handbook of child psychology*, 4th ed., Vols. I–IV. New York: Wiley.

Nauta, W. J. H. (1958). Hippocampal projections and related neural pathways to the midbrain in the cat. *Brain, 81*, 319–340.

Nauta, W. J. H. (1971). The problem of the frontal lobe: A reinterpretation. *Journal of Psychiatric Research, 8*, 167–187.

Nauta, W. J. H., and Domesick, V. B. (1978). Crossroads of limbic and striatal circuitry

Hypothalamonigral connections. In K. E. Livingston and O. Hornykiewicz (Eds.), *Limbic mechanisms* (pp. 75–94). New York: Plenum.

Nauta, W. J. H., and Domesick, V. B. (1980). Neural associations of the limbic system. In A. Beckman (Ed.), *Neural substrates of behavior* (pp. 173–206). New York: Spectrum.

Nauta, W. J. H., and Mehler, W. R. (1966). Projections of the lentiform nucleus in the monkey. *Brain Research, 1*, 3–42.

Nicholi, A. M. (Ed.) (1978). *The Harvard Guide to Modern Psychiatry*. Cambridge, MA: Harvard University Press.

Ono, J., Senba, E., Okada, S., Abe, J., Futagi, Y., Shimizu, H., Sugita, T., Hashimoto, S., and Yabuuchi, H. (1982). A case report of congenital hypomyelination. *European Journal of Pediatrics, 138*, 265–270.

Osofsky, J. D. (1979). *Handbook of infant development*. New York: Wiley-Interscience

Oster, H. (1978). Facial expression and affect development. In M. Lewis and L. A. Rosenblum (Eds.), *The development of affect*. New York: Plenum.

Papez, J. W. (1937). A proposed mechanism of emotion. *Archives of Neurology and Psychiatry*, 38, 725–743.

Parker, S., and Gibson, K. R. (1979). A model of the evolution of language and intelligence in early hominids. *The Behavioral and Brain Sciences, 2*, 367–407.

Peiper, A. (1963). *Cerebral function in infancy and childhood*, 3rd ed. New York: Consultants Bureau.

Peters, A., Palay, S. F., and Webster, H. deF. (1976). *The fine structure of the nervous system: The neurons and supporting cells*. Philadelphia: Saunders.

Plomin, R., and Rowe, D. C. (1978). Genes, environment and development of temperament in young human twins. In G. M. Burghardt and M. Bekoff (Eds.), *The development of behavior: Comparative and evolutionary aspects* (pp. 279–296). New York: Garland.

Prechtl, H., and Beintema, D. (1964). The neurological examination of the full-term newborn infant. *Little Club Clinics in Developmental Medicine No. 12*. London: Heinemann.

Purpura, D. P., and Reaser, G. P. (1974). *Methodological approaches to the study of brain maturation and its abnormalities*. Baltimore: University Park Press.

Rabinowicz, T. (1979). The differential maturation of the human cerebral cortex. In F. Falkner and J. M. Tanner (Eds.), *Human growth* (Vol. III pp. 97–123). New York: Plenum.

Raine, C. S. (1984a). Morphology of myelin and myelination. In P. Morell (Ed.), *Myelin*. New York: Plenum, pp. 1–50.

Raine, C. S. (1984b). The neuropathology of myelin diseases. In P. Morell (Ed.), *Myelin* (pp. 259–310). New York: Plenum.

Rasminsky, M. (1973). The effects of temperature on condition in demyelinated single nerve fibers. *Archives of Neurology (Chicago), 28*, 287–292.

Rasminsky, M. (1980). Ephaptic transmission between single nerve fibres in the spinal nerve roots of dystrophic mice. *Journal of Physiology (London), 305*, 151–156.

Rasminsky, M., and Sears, T. A. (1972). Internodal conduction in undissected demyelinated nerve fibers. *Journal of Physiology, 227*, 323–350.

Rheingold, H. (Ed.). (1963). *Maternal behavior in mammals*. New York: Wiley.

Ricciuti, H. N. (1974). Fear and the development of social attachments in the first year of life. In M. Lewis and L. A. Rosenblum (Eds.), *The origins of fear* (pp. 73–106). New York: Wiley.

Rinn, W. E. (1984). The neuropsychology of facial expression. A review of the neurological and psychological mechanisms for producing facial expressions. *Psychological Bulletin, 95*, 52–77.

Ritchie, J. M. (1984). Pathophysiology of conduction in demyelinated nerve fibers. In P. Morell (Ed.), *Myelin* 2nd ed. New York: Plenum.

Robson, K. S. (1967). The role of eye-to-eye contact in maternal-infant attachment. *Journal Child Psychology and Psychiatry, 8*, 13–25.

Robson, K. S., and Moss, H. A. (1970). Patterns and determinants of maternal attachment. *Journal of Pediatrics, 77*(6), 976–985.

Rorke, L. B., and Riggs, H. E. (1969). *Myelination of the brain in the newborn*. Philadelphia: Lippincott.

Rosenblum, L. A., and Alpert, S. (1974). Fear of strangers and attachment in monkeys. In M. Lewis and L. A. Rosenblum (Eds.), *The origins of fear* (pp. 165–190). New York: Wiley.

Rudolph, A. M., and Hoffman, J. I. E. (1982). *Pediatrics*, 17th ed. Norwalk, CT: Appleton Century-Crofts.

Sameroff, A. (Ed.). (1979). Organization and stability of newborn behavior: A commentary on the Brazelton Neonatal Behavioral Assessment Scale. *Monographs of the Society for Research in Child Development, 43*, 5–6 (Serial No. 177).

Sanneck, R. (1975). Training induced myelination in peripheral nerves of the rat. *Journal of Physiology, London, 244*, 7P.

Samorajski, T., and Rolsten, C. (1975). Nerve fiber hypertrophy in posterior tibial nerves of mice in response to voluntary running activity during aging. *Journal of Comparative Neurology, 159*, 533–558.

Scott, J. P. (1962). Critical periods in behavioral development. *Science, 138*, 949–958.

Sidman, R. L., and O'Gorman, S. V. (1981). Cellular interactions in Schwann cell development. In V. M. Riccardi and J. J. Mulvihill (Eds.), *Advances in neurology*, Vol. 29. *Neurofibromatosis (von Recklinghausen Disease)* (pp. 213–236). New York: Raven.

Sluckin, W. (1970). *Early learning in man and animal*. Cambridge: Schenkman.

Smith, K. J., Blakemore, W. F., and McDonald, W. I. (1981). The restoration of conduction by central remyelination. *Brain, 104*, 383–404.

Spitz, R. A., and Wolf, K. M. (1946). The smiling response: A contribution to the ontogenesis of social relations. *Genetic Psychology Monographs, 34*, 57–125.

Spock, B. (1976). *Baby and child care*, 4th ed. New York: Pocket Books.

Sroufe, L. A., and Waters, E. (1976). The ontogenesis of smiling and laughter: A perspective on the organization of development in infancy. *Psychological Review, 83*(3), 173–189.

Stern, D. (1974). Mother and infant at play: The dyadic interaction involving facial, vocal, and gaze behaviors. In M. Lewis and L. A. Rosenblum (Eds.), *The effect of the infant on its caregiver* (pp. 187–214). New York: Wiley.

Stern, D., and Gibbon, J. (1977). Temporal expectancies of social behaviors in mother-infant play. In E. Thoman (Ed.), *The origins of the infant's responsiveness* (pp. 409–430). New York: Lawrence Erlbaum.

Super, C. S. (1981). Behavioral development in infancy. In R. H. Munroe, R. L.

Munroe, and B. B. Whiting (Eds), *Handbook of cross-cultural human development* (pp. 181–270). New York: Garland.

Super, C., Kagan, J., Morrison, F., Haith, M., and Weiffenbach, J. (1972). Discrepancy and attention in the five-month infant. *Genetic Psychology Monographs, 85*, 305–331.

Svejda, M. J., Pannabecker, B. J., and Emde, R. (1982). Parent-to-infant attachment: A critique of the early "bonding" model. In R. N. Emde and R. J. Harmon (Eds.), *The development of attachment and affiliative systems* (pp. 83–94). New York: Plenum.

Tauber, H., Waehneldt, T. V., and Neuhoff, V. (1980). Myelination in rabbit optic nerves is accelerated by artificial eye opening. *Neuroscience Letters, 16*, 235–238.

Tennes, K. H., and Lampl, E. E. (1964). Stranger and separation anxiety in infancy. *Journal of Nervous and Mental Disorders, 139*, 247–254.

Thompson, J. (1941). Development of facial expression of emotion in blind and seeing children. *Archives of Psychology, 264*, 1–47.

Tilney, F., and Casamajor, L. (1924). Myelinogenesis as applied to the study of behavior. *Archives of Neurology and Psychiatry, 12*, 1–66.

Triandis, H. L., and Lambert, W. W. (1980–1981). *Handbook of cross-cultural psychology*, Vols. I–VI. Boston: Allyn and Bacon.

Traugott, U., and Raine, C. S. (1984). The neurology of myelin diseases. In P. Morell (Ed.), *Myelin*, 2nd ed. (pp. 311–336). New York: Plenum.

Valenstein, E. S. (1973) *Brain control*. New York: Wiley.

Van Hooff, J. A. R. A. M. (1972). A comparative approach to the phylogeny of laughter and smiling. In R. A. Hinde (Ed.), *Non-verbal communication* (pp. 209–242). Cambridge: Cambridge University Press.

Warren, N. (1972). African infant precocity. *Psychological Bulletin, 78*, 353–367.

Watson, J. S. (1972). Smiling, cooing and "the game." *Merrill-Palmer Quarterly, 4*, 323–339.

Watson, J. S., and Ramey, C. P. (1972). Reactions to response contingent stimulation in early infancy. *Merrill-Palmer Quarterly, 18*, 219–228.

Waxman, S. G. (1977). Conduction in myelinated, unmyelinated and demyelinated fibers. *Archives of Neurology, 34*, 585–589.

Waxman, S. G. (1982). Membranes, myelin, and the pathophysiology of multiple sclerosis. *New England Journal of Medicine, 306*, 1529–33.

Wendell-Smith, C. P. (1964). Effects of light deprivation on the postnatal development of the optic nerve. *Nature (London), 204*, 707.

Werner, E. E. (1972). Infants around the world: Cross-cultural studies of psychomotor development from birth to two years. *Journal of Cross-Cultural Psychology, 3*, 111–134.

Werner, E. E. (1979). *Cross-cultural child development*. Monterrey, CA: Brooks/Cole.

Whiting, B., and Whiting, J. (1975). *Children of six cultures*. Cambridge, MA: Harvard University Press.

Wiesel, T., and Hubel, D. (1965). Extent of recovery from the effects of visual deprivation in kittens. *Journal of Neurophysiology, 28*, 1060–1072.

Windle, W. F., Fish, M. W., and O'Donell, J. E. (1934). Myelogeny of the cat as related to development of fiber tracts and prenatal behavior patterns. *Journal of Comparative Neurology, 59*, 139–165.

Wolff, P. H. (1963). Observations of the early development of smiling. In B. M. Foss (Ed.), *Determinants of infant behavior II* (pp. 113–134). London: Methuen

Yakovlev, P. I. (1962). Morphological criteria of growth and maturation of the nervous system in man. *Mental Retardation, 39*, 3–46.

Yakovlev, P. I. (1970). Whole brain serial histological sections. In C. G. Tedeschi (Ed.), *Neuropathology: Methods and diagnosis* (371–378). Boston: Little, Brown.

Yakovlev, P. I., and Lecours, A. R. (1967). The myelogenetic cycles of regional maturation of the brain. In A. Minkowski (Ed.), *Regional development of the brain in early life* (pp. 3–70). Oxford: Blackwell Scientific.

Zelazo, P., Zelazo, N., and Kolb, S. (1972). "Walking" in the newborn. *Science, 176*, 314–315.